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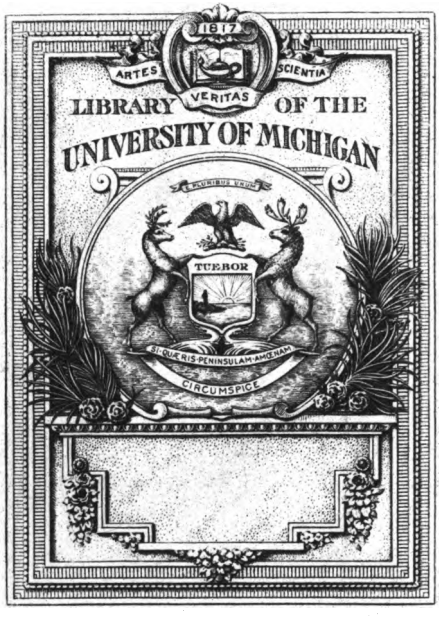

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TRANSACTIONS OF THE

SECTION ON

Diseases of Children

of the
American Medical Association
at the Sixty-Second Annual
Session, held at Los Angeles,
Cal., June 27 to 30, 1911

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PROCEEDINGS OF THE SESSION

Tuesday, June 27—Afternoon

The meeting was called to order at 2:20 p. m. by the Chairman, Dr. S. M. Hamill, Philadelphia.

In the absence of Dr. E. E. Graham, Philadelphia, member of the Executive Committee, the Chairman appointed Dr. W. W. Butterworth to fill the vacancy.

Dr. S. M. Hamill, Philadelphia, read the Chairman's Address on "The Opportunities of the Pediatrist in the Prevention of Death and Diseases in Early Life."

Dr. Richard R. Smith, Grand Rapids, Mich., read a paper on "A Study of Children in Reference to Enteroptosis." Discussed by Drs. W. J. Butler, Chicago, and I. A. Abt, Chicago.

On motion, Dr. Frank Allin, of Chicago, was granted permission to read a paper not on the program.

Dr. Thomas S. Southworth, New York, read a paper on "The Institutional Care of Young Infants." Discussed by Drs. Gustavus Eliot, New Haven; J. W. Cokenower, Des Moines; Langley Porter, San Francisco; I. A. Abt, Chicago; W. W. Butterworth, New Orleans; T. C. McCleave, Berkeley, Cal.; R. B. Tubbs, Council Bluffs; Jessie M. McGavin, Portland; W. V. C. Francis, Los Angeles, and T. S. Southworth.

Dr. Frank Allin, Chicago, read a paper on "The Baby Tents in Chicago." Discussed by Drs. J. P. Sedgwick, Minneapolis; May Michaels, Chicago, and Frank Allin.

Wednesday, June 28—Morning

Dr. I. A. Abt, Chicago, read a paper on "The Influence of Summer Heat on the Production of Gastro-Intestinal Diseases of Infants." Discussed by Drs. John W. Foss, Phoenix, Ariz.; C. L. Magee, Los Angeles; M. A. Weems, Columbia, Tex.; W. V. C. Francis, Los Angeles; Frank P. Gengenbach, Denver; Frances M. Allen, San Diego; W. W. Butterworth, New Orleans; L. T. Royster, Norfolk, Va., and I. A. Abt.

Dr. W. W. Butterworth, New Orleans, read a paper on "A Statistical Investigation Relative to the Influence of Various Methods of Infant-Feeding on the Morbidity and Mortality of Infants, Occurring in New Orleans, from August 1, 1909, to July 31, 1910."

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Dr. F. C. Neff, Kansas City, Mo., read a paper on "Recent Experiences in the Artificial Feeding on 100 Infants During the First Three Months of Life."

These two papers were discussed by Drs. Thomas S. Southworth, New York; J. R. McLaren, Oakland; M. Dranga, San Diego; Langley Porter, San Francisco; J. P. Sedgwick, Minneapolis; R. B. Tubbs, Council Bluffs; A. L. Tilton, Kingman, Ariz.; L. R. DeBuys, New Orleans; I. A. Abt, Chicago; L. T. Royster, Norfolk; W. V. C. Francis, Los Angeles; C. L. Magee, Los Angeles; W. W. Butterworth and F. C. Neff.

Drs. J. P. Sedgwick and F. W. Schultz, Minneapolis, read a paper on "Relationship of Gastric to Pancreatic Fat Digestion in Infants." Discussed by Dr. S. M. Hamill.

Wednesday, June 28—Afternoon

Dr. Philip King Brown, San Francisco, read a paper on "The Neurotic Basis of Juvenile Delinquency, Based on Some Special Cases of the San Francisco Juvenile Court." Discussed by Drs. John A. Colliver, Los Angeles; L. T. Royster, Norfolk, Va.; F. R. Branham, San Diego, Cal.; D. S. Hanson, Ohio, and P. K. Brown.

Dr. Langley Porter, San Francisco, read a paper on "A Consideration of Obstructions at the Pylorus Occurring in Infants." Discussed by Drs. W. W. Butterworth, New Orleans, and L. Porter.

Dr. L. Porter read a paper written by Dr. E. C. Fleischner, San Francisco, on "The Relation of Meat Ingestion to Indicanuria in Childhood." Discussed by Dr. J. P. Sedgwick, Minneapolis, and Dr. Porter.

As Dr. Butterworth desired to be relieved from duty on the Executive Committee, the Chairman appointed Dr. Frank P. Gengenbach, Denver, to fill the vacancy.

Thursday, June 29—Morning

Dr. J. W. Cokenower, Des Moines, Ia., read a paper on "Rachitis in Its Early Stages." Discussed by Dr. Elizabeth Follansbee, Los Angeles, and Dr. Cokenower.

The Executive Committee presented the following nominations as a majority report: Chairman, Dr. I. A. Abt, Chicago; Vice-Chairman, Dr. L. T. Royster, Norfolk, Va.; Secretary, Dr. J. P. Sedgwick, Minneapolis, and Delegate, Dr. William Hollopeter, Philadelphia. Moved by Dr. Southworth, and seconded, that the Secretary be empowered to cast the ballot of the Section for these officers.

It was moved and seconded that the minority be allowed to present a report. Motion was lost.

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The motion of Dr. Southworth was voted on and carried.

Dr. William J. Butler, Chicago, read a paper on "Report on Brain Cases in Children." Discussed by Dr. C. C. Rogers, Chicago, and Dr. Butler.

The Section adjourned at 10:15 to meet with the Section on Practice of Medicine in the afternoon.

THURSDAY, JUNE 29—AFTERNOON

The following papers were read at a Symposium on Poliomyelitis—a joint session with the Section on Practice of Medicine.

Drs. Samuel G. Dixon, Harrisburg, Pa., and H. T. Karsner, Philadelphia, presented a paper on "Epidemiologic and Etiologic Studies of Acute Poliomyelitis in Pennsylvania."

Drs. R. W. Lovett and M. W. Richardson, Boston, presented a paper on "Infantile Paralysis."

These papers were discussed by Dr. Philip King Brown.

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THE INSTITUTIONAL CARE OF YOUNG INFANTS

THOMAS S. SOUTHWORTH, M.D.
NEW YORK

A recent writer on "The Proper Management of Foundlings and Neglected Infants,"[1] has gone so far as to indicate his belief that the institutional method must give way entirely to the system of placing out in families. He says:

> The first step would be entirely away from institutional care, as the improved feeding-methods and personal hygiene have not had the favorable results expected while the infants are collected and retained in large numbers in one domicile. These old and futile efforts have been long and faithfully tried, and it is time that some other plans be instituted, if the mortality among this class of infants is to be materially reduced.

That the older methods in institutions for foundlings and young infants were productive of a shocking mortality among those under one year of age, has been acknowledged the world over. Nevertheless, reasons existed and still exist for the retention of the institution in some modified form. While some institutions to-day continue to pursue fatalistically the older methods, it must be admitted that others have made marked strides and are presenting very enviable results. But my conviction is that only a beginning has been made, and that far better results can be obtained. The purpose of this paper is, therefore, to call attention to the opportunities and obligations of institutions which receive and care for infants.

1. Chapin, Henry Dwight: Med. Rec., New York, Feb. 18, 1911.

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FACTORS IN INSTITUTIONAL MORTALITY

The factors which enter into the large mortality in foundling asylums and institutions are as follows:

1. Delay in transmission of the infants to the institution, with resulting disturbance of vital functions and of digestion.

2. Questionable initial care on entering the institution.

3. No wet-nurses, or too few wet-nurses, for premature and delicate babies.

4. Bottle-feeding from birth, or sudden weaning on reception in the institution.

5. Inadequate facilities (milk laboratories or diet kitchens) for preparing bottle-feedings.

6. Routine artificial feeding, with but slight individualization.

7. Insufficient nursing force, which precludes the normal personal attention to each infant.

8. Large wards with ready extension of infectious and contagious principles.

9. Overcrowding, with lack of air-space and of proper ventilation.

10. Insufficient provision for outdoor airings.

11. Inadequate facilities for the application of quarantine, in its broadest sense, to all sources of infection in infancy.

It may be remarked that I have given no prominent place to the requirement of clean milk and of methods intended both to keep it sweet and to limit bacterial growth. This is necessarily included in the proper preparation of artificial feedings, and its importance has been unduly exaggerated and attention thereby diverted from other measures of equal if not greater urgency.

Such degree of success as has been attained by the institutions which now show the more enviable records has come to them in proportion to their recognition of the adverse factors which I have enumerated.

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To put this reorganization of institutions for infants on a logical basis, we must not be content with the correction of isolated errors; we must go far enough back to discover the fundamental reasons for their abnormal mortalities. In this search, we may start from the premise that as a rule the lowest mortality obtains where infants receive not only the mother's breast-milk, but also her personal care in the home. It matters little how primitive the home, if there be competent breast-milk and individual care, the prospects of survival for an infant, healthy at birth, are excellent. Remove one of these factors, and the risk is greatly increased. We have daily cause to recognize this added risk when artificial feeding must be resorted to, even in a home where there is maternal care. Deprive the infant both of breast-feeding and of home care, and at once the outlook becomes an exceedingly serious one.

While institutional environment undoubtedly contributes its own special dangers, it is generally recognized that when breast-fed or bottle-fed infants in an institution are accompanied by their mothers, they do relatively better than infants correspondingly fed who have no maternal attention. Such success as the placing-out system has obtained is dependent in very considerable part on restoring to the infant individualized care in the substitute home. Therefore, since breast-milk and motherly care in the home are the foremost factors in preventing early mortality, and experience has shown us that possibly either one of the two, but never both, can be dispensed with without incurring the greatest risk, it follows that just in proportion to the ability of the modern institution to furnish these to infants will be its success in preventing their deaths and rearing them as normal well-nourished children.

Nevertheless, it is just these two factors which are most lacking in the majority of institutions, and until they are supplied more adequately, it cannot be claimed

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that the limits of success in the institutional care of infants have been reached.

We may now return to the consideration of the special factors which call for readjustment.

Transfer to the Institution.—The institutional life of an infant is usually preceded by some period of stress in the home from which it comes. This may be due to the illegitimate birth of the infant, the death of the mother, her serious illness, or merely to the mental anxiety associated with increasing poverty or the breaking up of the home. These instances will suffice to indicate that the infant, whether breast-fed or bottle-fed, has usually suffered more than ordinary deprivation and neglect. Then follows the transportation of the infant, often unsuitably clothed, to the institution. Possibly the first application is made with the infant at the police station, the office of some charitable organization or some general hospital. This may involve long waits or another journey. If the baby is taken in at some temporary receiving station, misguided sympathy gives the infant some unsuitable milk mixture to still its cries. Eventually it reaches the institution, suffering from unwonted exposure to heat or cold, fatigued, and probably with a disturbed digestion. In short, infants on reaching the institution are so rarely in normal condition that I recall one institution in which it was the routine practice to enter the condition of all such infants as "hopeless," in order to avoid criticism in the event of their later demise. The partial solution of this problem should lie in better arrangements at all charitable centers and receiving stations for prompt attention to such infants, and for immediate transfer to their proper destination.

Reception and Attention at the Institution.—When the child is received into the ward, the probable previous experiences should be borne in mind. If it has been exposed to cold, it should be given a warm bath to restore the circulation, enveloped in warm clothing and placed

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in a warm bed. Every institution receiving such infants should be equipped with suitable appliances for maintaining the temperature of feeble and exhausted infants when this is below normal. Premature infants are especially vulnerable. The incubator has gone out of fashion. It easily got out of order, and fresh air was rarely supplied in sufficient quantity. Its place has been taken by enveloping suits of cotton-wool and cheese-cloth, and by padded baskets in which the body heat is maintained by hot-water bottles, or—much better—by electric heating pads. A "warm room," in which abundant fresh air is heated uniformly to 80 F. or thereabouts, will in time come to be a necessity in every well-appointed institution for infants.

Nourishment.—The nourishment of the infant following its reception is a matter of great importance. Bearing in mind its previous experiences, this is a moment for caution. If the child has been previously breast-fed, this is a particularly inauspicious time for beginning total artificial feeding. If the infant has been fed artificially, in an ignorant or haphazard fashion, the digestion is probably more than ordinarily disturbed. Rest and fluid in the form of boiled water are more advisable than food, unless this be very simple—barley water or whey. Exact information as to the infant's previous food is rarely obtainable. A mild laxative to clear the digestive tract of the injudicious offerings of solicitous friends or strangers is a wise preliminary to the beginning of a cautious line of feeding.

Temporary Wet-Nursing.—For many of these infants, temporary wet-nursing should be provided. No other measure offers an equal chance of restoring digestive balance. While the sudden weaning which takes place on the transfer of an infant to the hospital is so often the beginning of a more or less prolonged period of malnutrition ending in death, wet-nursing for a few days will permit the gradual introduction of bottle feeding, and is not only more rational, but more fre-

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quently attended with success. Whatever may be said of the ethics of wet-nursing in private practice whereby the life of the wet-nurse's child is often sacrificed in order to save that of the foster-child, wet-nursing in the institution has neither this drawback nor is it impracticable. It is simply a question of dollars and cents.

Breast-milk is purchasable. Institutions which undertake to confine and shelter prospective mothers without pay should very properly exact an agreement that the women will nurse another baby besides their own, as a partial return for what they themselves receive. Homeless mothers can be found who are glad to accept the shelter of the hospital while earning something for the future, through the sale of part of their breast-milk. Circumstances should determine the manner of payment. Exceptionally willing nurses may be paid a lump sum per month. For others, the transaction should be on the strictly commercial basis of so much per ounce for the breast-milk furnished. This plan, followed at the Säuglingsheim at Dresden and elsewhere, at once assures the cooperation of the wet-nurse. She is more ready to confine herself to a proper milk-making diet, since the larger flow of milk is to her advantage. The quantity taken from the breast is easily determined by weighing the baby before and after nursing. It is stipulated at the start that she is to nurse any baby assigned to her. To ensure this being done, milk, when drawn from the breast and fed from the bottle, is paid for at a somewhat lower rate.

By furnishing temporary wet-nursing, it is possible for the institution to compensate its infant inmates at crucial junctures for the serious loss of maternal nursing.

Artificial Feeding.—Even with a reasonable provision for wet-nursing, the majority of infants in any institution must be fed artificially. This is far from being a simple matter. It is generally admitted by all who have had any experience in this direction that it is much easier to feed artificially a group of infants who are

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cared for in their homes, even though the homes be poor ones, than to feed successfully a similar group in an institution ward. Furthermore, if institution infants are fed on condensed milk, simple dilutions of cow's milk with sugar and water, or even with a few inelastic percentage formulas of what is commonly called "modified milk," a considerable proportion of the results will be unsatisfactory, and the deaths, especially among very young infants, numerous. Every institution should possess a supply of clean cow's milk, and a milk laboratory or kitchen thoroughly equipped and sufficiently elastic to permit of individualization of the infants in their feeding, and the employment, as required, of every modern scientific aid. Only with such aids, backed by experience in their application, can the mortality of artificially fed infants in institution or hospital wards be reduced to a reasonable minimum. Such a milk laboratory and its maintenance cost money, but it is well expended for the results are to be measured in human lives.

Even with these facilities conscientiously applied, however, there are certain infants who, from failure to absorb their carefully adjusted food, or frequently also as the result of intercurrent disease, lose steadily in weight. Under such circumstances, in private practice the resort is to a wet-nurse with a reasonable prospect of staying the decline and reestablishing normal digestion and absorption. The institution or hospital infant has a right to similar treatment before it is deemed hopeless and condemned to die on a regimen which has already proved inefficacious. Wet-nurses employed by the institution have here another important field of usefulness. It is not necessary that such wet-nursing should be continued indefinitely. When, with gain in weight and restored digestive functions, the infant has been lifted out of the danger zone, artificial feeding may be resumed. This is at first partial, enabling the wet-nurse to give more of her milk to other needy infants, and in

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time it is made total. A few nursings a day often enable an infant to assimilate more successfully the bottle-feedings which furnish the most of its nourishment.

We have now pointed out the lines along which the institution may progress in compensating infants for the loss of maternal nursing, and may turn to the ways in which the loss of individual care and protection in the home may be more properly compensated.

Large Wards.—Unquestionably, the building of the large wards which prevail almost universally in hospitals and institutions had its origin in the desire to care for the largest possible number of patients, with the aid of the smallest possible number of nurses or attendants. While the evils of this policy can, where adults are concerned, be largely overcome by increase of the nursing staff, the noxious influences of the large ward on children are too numerous and too insidious to be met solely by such measures. The late Mark Twain said that in the home "one baby was a riot, twins an insurrection, and triplets a full-blown revolution." The average mother or nurse in the home would be considered in danger of overwork in giving proper care to three infants. Many institution wards, however, are constructed to house from sixteen to twenty-four infants, while during the day three attendants, of varying degrees of experience, and one at night, would be considered a liberal provision, even when the ward contained a number of sick infants. Such disproportion, even if it allows for the more necessary services, certainly precludes any of the refinements of motherly solicitude, which, in some indefinable way, aid in keeping a baby in good condition.

Overcrowding.—Aside from the lessened attention given each infant, the overcrowding of wards has unquestionably a deleterious effect on young infants. Because infants are small and their cribs small, they are often allowed less air-space than the adult. Just the reverse of this should obtain. Oxidation is much greater during

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the normal rapid growth of infancy. Free ventilation is therefore imperative. This calls for properly screened windows open at all seasons both day and night. Fresh and not overheated air, now demanded in the treatment of disease, has no less place in the welfare of infants (not premature) who are nominally well. Even with careful attention to ventilation, it is a common experience to find that when a certain number in each ward is overstepped, all the bottle-fed infants begin to do less well.

Probably the greatest menace to the infant in the large ward is infection. The element of added risk, when the infant leaves the comparative seclusion of its own home and enters the populous ward, is scarcely computable. Suffice it to say that the chance of infection is multiplied by the number of other inmates in the ward, since, with a few exceptions, the infections to which it is liable come most commonly from other children, and not from adults.

In speaking of infections and contagions, it is natural that we think first of those popularly associated with infancy and childhood. Scarlet fever and mumps play less part among the very young. Diphtheria is somewhat better controlled than formerly, through antitoxin immunization, but repeatedly breaks out in unexpected places through the agency, probably, of unrecognized bacillus carriers. The exclusion of specific vaginitis calls for constant vigilance. Whooping-cough and measles spread easily, defy ordinary formal quarantine, and cause, both directly and indirectly, many deaths. Illnesses which are regarded lightly in the home assume a more serious import when they occur in a ward of bottle-fed infants who are with difficulty maintaining their weights or even making moderately satisfactory gains. Chicken-pox stops gains and claims some victims among the feeble. Thrush ("sprue") is a serious and often fatal disease in such a group, costing marasmic infants a pound or more of weight, a loss which many cannot survive.

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This by no means completes the list of dangerous infections. I am inclined to the belief that two other groups of infections actually cost more lives in institutions having large wards. I refer to the communicable infections of the gastro-intestinal tract which predominate in the summer months, and the communicable diseases of the respiratory tract, which occur chiefly, but not exclusively, in the winter months. Whatever improvement there may have been in other directions, little attention has thus far been paid to isolation, quarantine and other preventive measures for these conditions. There is growing evidence that ward epidemics of intestinal disturbance of varying degrees of severity travel from patient to patient, or arise from common causes not traceable to the milk. Ward epidemics of nasopharyngeal disease are easily recognizable, carrying in their train otitis media, pneumonia and cervical abscess. Isolation in its broader application is not only not practiced, but cases of pneumonia, frankly infectious, are rarely removed from the general wards in which they appear, unless it be for treatment by cold air or in special medical wards. It may be claimed that it is practically impossible to isolate all cases of potential infection in our institutions. If, however, it be really impossible to take measures for the limitation of avoidable illness, the main fault lies with the construction and arrangement of the building. If infections of all kinds are to be limited at the outset, the large ward with many inmates must go.

The construction of new institutions and hospitals for infants must therefore include, primarily, smaller wards, preferably containing four, but not more than six beds. Each ward should have its own toilet and bathing-room, and a smaller room opening out of it, in which any patient showing doubtful symptoms may be placed by the nurse. Each ward should also have a section of veranda on which the infants may be placed in lined and padded baskets on all favorable days. The

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influence of such outdoor airings on the health, color, digestion and growth of infants has been demonstrated. Each ward should have its own semipermanent nurse or nurses. A more intimate relation will thus be established between the nurse and her charges, a more definite responsibility felt for their welfare, and an honorable pride and rivalry in their progress will be stimulated. With such arrangements, the composite element of "hospitalism" should be largely eliminated. If infants and young children of different ages are assigned to each small ward, conditions will more nearly approximate the home and more personal attention will be secured for the infants. Where training-schools for nursery-maids exist, the training acquired will fit them more suitably than at present to meet their responsibilities when they enter private service.

I am no opponent of the placing-out system, but until we recognize the fundamental defects of the older types of institutions, and cutting loose from tradition and error, rectify them, calling nothing impossible or impracticable until it has been tested under favorable conditions, we certainly are not justified in saying that the institutional care of infants is a failure. The future institution, built and organized to avoid present-day mistakes, should, in view of the showing made by the best of the old-type institutions, attain a proportionately greater measure of success. Then, as now, the most favorable results will be secured, not by the abandonment of either, but from organized cooperation of both systems.

807 Madison Avenue.

ABSTRACT OF DISCUSSION

Dr. Gustavus Eliot, New Haven, Conn.: The most wonderful cures I have known of in children ill with digestive and diarrheal troubles in summer have followed the carrying of children from the city to the country. This leads me to emphasize the point that such institutions for the care of children should be removed from the city and erected in the country. The buildings should not be the elegant three- or

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four-story buildings such as are often seen in the cities, but they should be low buildings so constructed that after a few years' use they may be destroyed and clean new buildings take their place. The location of such an institution in the country also gives an opportunity for improvement in the treatment of these children in regard to their food. They are fed to a great extent on cows' milk, and if in the country, they can receive milk that is absolutely fresh and clean. In the feeding of these children, fresh milk twice a day should be used instead of the milk that is now used, which is twenty-four to forty-eight hours old when it reaches the consumer. These two things, country air and fresh, clean milk, are most important factors in the treatment of infants deprived of a mother's care.

Dr. J. W. Cokenower, Des Moines, Iowa: In twenty years' work in the Iowa Children's Home, in the admission of babies, much trouble has been caused us because we could not get a complete history. We have found that in nearly all such institutions nine times out of ten the infectious and contagious diseases have arisen from receiving diseased patients. This we have partially overcome. One must look after the excretions and secretions and deal with them in any way that will bring the best results. After that is done, no routine method will fill the bill. Individual cases require individual feeding. We use barley water, and sometimes have employed prepared foods and malted milk, but it is the exception and not the rule when we have been able to use them advantageously. But cows' milk has been our chief dependence and we try to keep that absolutely clean, and have much better results. Often we have children brought to us in such a condition that we are unable to do good work. The stomach absolutely refuses to retain anything, either food or medicine, and there is such a generally relaxed condition that we cannot give it by the rectum; then as a last resort we have used quinin and olive oil, making an inunction and wrapping them in cotton and oiled silk. In that way we feed and medicate them until we have given the digestive organs a rest and can feed them otherwise, and in this way have saved several children that I believe we would otherwise have lost.

Dr. Langley Porter, San Francisco: I think no one could have presented a more forcible argument against institutions than Dr. Southworth in the paper he has just read. I believe that twenty years from to-day no one in this Section of the American Medical Association will make a plea for the retention of the institution. In San Francisco we have persuaded the Children's Agency of the Associated Charities to do away with their foundling asylum and to board out the children, with the result that the death-rate has been reduced from 40 to less than 2 per cent. But the care of the babies in the institutions is really a very small part of the question. The

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real problem is to educate the public in caring for children. When a baby is boarded out, the result is that the older children in the family are being trained to the proper care of children, and that is the first and greatest advantage in the boarding-out system. A second advantage is the decrease in the death-rate that always follows. The boarding-out system is of economic value, also, for the reason that in the fixed charges of such an institution, as Dr. Southworth has described, we tie up a large amount of money and also pay out much money to people for the care of these children. If you were to divide this money among the many needy families, allowing them to care for these children, you would get better value for the money than if it were tied up in capital. I believe that before many years have passed Dr. Southworth will be as ready to condemn institutions as any of us.

Dr. I. A. Abt, Chicago: I agree for the most part with the last speaker, though I think it will be many years before we can abolish infant hospitals and asylums. Some place must be provided where we can send babies that are destitute and require immediate care. I think the infants' asylum or hospital is valuable just in proportion to what is put into it. It is of value just in proportion to the amount of soul and brains put into it. If you want results you must have a technic that provides for asepsis and cleanliness, good food and good care. This must be carried out with determination, and in the end you will save a goodly proportion of your children. In boarding-out cases, the result depends very largely on the character of the women who receive the patients. I have seen both ways tried. In the boarding-out system very often it is a mercenary matter with the women who take the babies. So far as wet-nurses are concerned, it has been said that an infants' hospital has no more right to exist without the wet-nurse than the adults' hospital without an operating room. I think that is true. If you desire to reduce the infant mortality, wet-nurses are absolutely necessary, especially in the summer when the mortality is high. In our own hospital in Chicago we have as many wet-nurses as we need. We found we could train these women to produce large quantities of milk. They milk the breasts and produce several quarts per day. As to the prevention of infection I think very often our technic needs to be perfected. It seems to me we are not careful enough about our hands, and sometimes the nurses infect a child. It all comes to this, that we must educate the educators, particularly physicians and nurses. Mothers and nurses should be taught how easy it is to carry infection from one child to another; not only vulvovaginitis, but a host of other diseases. Each baby ought to have its own individual property, its own toilet articles, its own powder box, even its own thermometer. Another important thing is the feeding of children when they first come to the hospital. I think we have

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gone too far in the matter of starvation of babies. I think that when a baby is brought in pinched, blue and cold and is given barley water for a prolonged period his weight-curve goes down and if he does not actually starve, at least he falls a ready victim to infection. I believe that starvation is an important element not only in causing the immediate death of the infant but indirectly predisposes to death from infection. Another matter of importance is the question of cathartics. I am convinced that many of these babies are physicked sick. If they have had a cathartic on admission and the intestinal tract is clean, what is the sense in giving them continued carthartics? I think the matter of starvation and the matter of over-catharsis should be emphasized, because at present they are often applied in a routine way without due reference to the condition of the patient.

Dr. T. C. McCleave, Berkeley, Cal.: Dr. Porter omitted to mention a very important feature of the work in San Francisco. This work was undertaken about two years ago. The mortality under the old system was about 40 per cent. and during the first year under the boarding-out system it dropped to 20 per cent. Then, an organization in the city making it possible for these children to be given certified milk, the mortality dropped in the second year to what it now is, about 2 per cent. Dr. Southworth spoke of clean milk. I think the meaning of that word "clean" is not always clear. In the feeding of infants and also of adults sick with various diseases, we know the wonderful change that results from putting the individual on certified milk. Therefore I want to urge on you particularly that you turn more and more to the use of certified milk. We have in California a strong organization of milk commissions, and we have a constantly increasing demand for certified milk. We are engaged in a vigorous propaganda among the laity for certified milk, as well as among the profession, but it is unfortunately true that it is often easier to get a response from the layman than from the physician.

Dr. Jessie McGavin, Portland, Ore.: I do think that one of the most important things is to see that the nurse carries out the physician's orders. It means a constant looking after the nurses until they are thoroughly trained, and when the nurse once understands that you are a crank, and that your orders must be carried out, you will save more of your babies. I lost one baby by neglect on the part of the nurse in the matter of feeding. In regard to contagious cases, in one instance I found a contagious hospital and a general hospital adjoining and the place simply swarming with flies. We need to educate the hospitals in these conditions. As to pure milk, in Portland we have had one epidemic with a very high mortality as a result of dirty milk. All physicians were losing

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cases. We have had a fight for pure milk and as a result our mortality is lessened.

Dr. T. S. Southworth, New York: Perhaps in some sections of the country the boarding-out system can be employed exclusively, but as I see conditions in the East it will be impossible for a long time to come. The sectarian organizations will be loth to give up their institutions. We still have a considerable field for improvement in our institutions and what I have said applies also largely to babies' hospitals. I recently saw a statement to the effect that being a baby should be classed as an extra hazardous occupation, and it is very true. I did not refer to the glass partitions, for such partitions are valuable just so far as you can depend on your nurses. The glass partition is merely a danger sign board. Speaking of food, we have certainly need of milk which is clean. However, we must not only have suitable milk, but it must be used in the right way. There has been too much talk in this country about the saving of life by pasteurization alone. As to the limitation of infections, reference has been made to the danger of the patient's bringing in infectious diseases on admission. I admit this is a serious matter, but a great deal of infection is introduced into the institution by the visits of parents or relatives. The outbreaks of the ordinary infectious diseases in the wards are directly proportionate to the number of friends allowed to come to see the children. Often a parent will come directly from the bed-side of a child sick with scarlet fever or other infectious disease.

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INFLUENCE OF SUMMER HEAT ON DIARRHEAL DISEASES

I. A. ABT, M.D.
CHICAGO

A first glance at infant-mortality tables demonstrates the fact that the greatest death-rate occurs from the gastro-intestinal diseases during summer. Dr. W. D. Booker, in the presidential address before the American Pediatric Society in 1901, reviewed the early literature on this subject. Dr. Booker says that the records show that in about the middle of the eighteenth century there occurred along the Atlantic coast of North America a serious disease characterized especially by vomiting and diarrhea and was confined almost exclusively to children in the first two years of life. Its incidence was limited almost to the summer months and it appeared each year in an epidemic form, with such regularity that it was looked for as an annual visitor. In 1777, Benjamin Rush read a paper entitled "An Inquiry Into the Cause and Care of Cholera Infantum." He gave a description of the stools, etiology and treatment of the disease. He denied the etiologic importance of dentition, worms and summer fruits, and he thought that the disease was similar to cholera and remitting fever in adults. He did not attempt to explain any relation of the disease to atmospheric heat, but contented himself by saying that the disease seldom occurred in Philadelphia until the middle of June and generally continued until the middle of September, and that its intensity and duration were always proportionate to the heat of the weather. The first indication for treatment, he thought,

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was to evacuate the stomach by emetics, followed by an abundance of water or tea, or an infusion of blueberries. He also suggested enemas, plasters to the abdomen, and internally he recommended port wine or white wine. The best results were obtained when a little water was mixed with the wine. He considered baths very important, and he said his principal dependence had been placed for many years on country air. As prophylactic treatment he recommended, (1) a daily cold bath, (2) clothing which was appropriate to the conditions of the weather, (3) removal of the children from the city to the country. Rush says: "I have never known but one instance of a child being affected with this disease, who has been carried into the country in order to avoid it."

It was thought at this time that the disease was peculiar to America as, apparently, the condition had not been described in other countries. It was thought to be due to the character of our climate, to the severity, frequency and suddenness of the change of temperature and to the sudden transition from winter to summer without the intervention of a gradually opening spring.

The mode of action of atmospheric heat in causing cholera infantum was the subject of controversy and various theories were advanced in explanation of it. These were for the most part based on the prevailing conception of the existence of a cutaneo-hepatic sympathy. By some, the heat of summer was supposed to exhaust the tone of the skin or to disturb its healthy action and thus determine an undue quantity of blood into the abdominal viscera, causing a congestion of these organs. Booker goes on to say that others thought that the effect of protracted heat led to a disturbance of the digestive organs because of some deranged condition of the exterior surface of the body. In this way the tone and energy of the stomach became impaired and consequently the system became more accessible to impressions exercised by external agents and its resistance to them became diminished.

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So far as the geographical distribution of the disease is concerned, it is noted that in the extreme north or south, the sick-rate and the death-rate are low. In North America, the middle and western states contribute the largest numbers in the mortality figures. As one approaches Canada, the numbers diminish; in the South and Southwest, we are told, the disease is less common. It is rare in the tropics, as has been observed in Brazil and in the tropical and subtropical regions of Asia and Africa. The same points in vital statistics apply to European countries. The summer diarrheal diseases occur most frequently in the large centers of population, particularly the industrial centers and in those regions where artificial feeding is commonly employed, and where the climatic conditions are such that the summers are relatively hot and the winters cold and severe.

The older American physicians, particularly Rush, thought that the hot weather was the most important factor in the etiology. He advised taking the child from the city to the country. In the course of time, other writers thought that the hot weather alone was an insufficient cause. Some said that the summer diarrhea of infants was a species of Asiatic cholera; others, that it was a form of malaria. In America, some writers considered it akin to yellow fever and others thought it was due to an indefinite miasm. Virchow thought that the condition stood in some relation to the water-supply and drainage, or to the surface water, adopting the view Pettenkofer applied to typhoid fever. In short, the older views as to the etiology of summer cholera considered that the disease was due to exogenous agents, the action of which was favored by the external heat, the latter acting either directly or through the food.

When the bacteriologic era began the attempt was made to find specific microorganisms. Escherich differentiated an exogenous infection of the intestines, the so-called streptococcus enteritis, and an endogenous infection, a process which ran its course as an auto-

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infection, or an infection of the chyme. Escherich thought that the disease might be produced by infected cow's milk, microorganisms gaining access to the milk shortly after milking, or that endogenous decomposition occurred as the result of already sick or enfeebled children receiving such milk. In the intestinal canal, irritating organic acids were believed to be produced, or alkaloidal products, ptomains, which produced diarrhea and nervous symptoms. No specific microorganism was found, but various forms, he thought, were capable of producing such a reaction. Other authors described epidemics of summer diarrhea, particularly in hospitals and asylums, and naturally thought that the epidemics were bacterial in origin. Baginsky, who failed to find a specific microorganism, came to the conclusion that the saprophytic bacteria in the milk were capable of producing poisonous peptones from the proteids. He thought that indol, phenol and ammonia gained access to the lymph and blood and produced toxemia. It is a well-known fact that bacteria multiply rapidly in milk during hot weather and it was thought that this was the sole cause. This led to sterilization and pasteurization of milk. Flugge and all the other authors who have described the poisonous effects of milk in producing summer cholera have compared this condition to meat-poisoning. But this comparison does not hold in all details. Meat-poisoning and sausage-poisoning affect all persons, whether they be children or adults, who have partaken of this food. It might be said that children are more susceptible than adults, though as a rule not all children who partake of supposedly infected milk fall ill. On the other hand, Vaughn has described epidemics of poisoning by ice-cream and cheese, affecting every person who partook of the food. They fell severely ill with vomiting, diarrhea, pain, weak, irregular pulse and great pallor or cyanosis. He isolated a poisonous substance which he called tyrotoxicon. No further

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argument need be advanced that the summer complaint is not similar to tyrotoxicon poisoning.

Czerny and Kellar believe that summer diarrheas are for the most part the result of exogenous fat-decomposition due to the presence of bacteria; they think that this condition is related to the heat of the summer only in so far as the increased number of microorganisms are rendered more active. But there are numerous objections to this theory. If it is true that bacterially acidified milk is toxic, why is buttermilk devoid of toxicity and why so acceptable, even to the sick infant?

In brief, it may be said:

1. The bacterial toxicity of milk is not proved.

2. On the other hand, a bacteria-laden milk is more likely to produce diarrhea than milk poor in bacteria.

3. In those cases in which a toxic product has been isolated from milk or meat, nearly all persons who took such food were rendered severely ill. An analogy of this condition with summer diarrhea is not tenable.

4. It is very probable that bacteria play a part in summer diarrhea, but they do not act on the milk before it is taken into the body (exogenous), but more likely cause fermentation after the food has been received in the gastro-intestinal tract (endogenous).

The bacterial explanation for the summer diarrhea is not sufficient. Why the excessively high mortality only during the hot weather? How may we explain the fact that the hotter the summer, the greater is the sick-rate and the death-rate among infants?

If during a hot day one examines the conditions under which the infants live in homes of the poor, one will be impressed by the fact that the temperature indoors exceeds the temperature outdoors; that at night, if the temperature outdoors has fallen considerably, the temperature indoors is considerably higher. The babies are frequently put to bed overclothed and sometimes excessively covered with bedding. If the infant's temperature be taken even before diarrhea has set in, he is frequently

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found to have 100 to 101 F. Many writers have looked on this condition as one of insolation or heat-stroke.

The interesting fact has been brought out that seven times as many artificially fed children, as breast-fed, die in summer. The bottle-baby is at greater peril in summer than at any other period of the year. No matter what theory may be advanced, the fact that breast-fed children show greater resistance and immunity than bottle-fed children cannot be denied. Those infants who have previously suffered from gastro-intestinal derangement, or even those who are convalescing from some alimentary disorder, are likely to show a hyperthermia on the advent of hot weather. This is undoubtedly due to the fact that the heat-regulating mechanism is much more unstable in children suffering from digestive disorders, and we may say that such children are predisposed to heat-stroke.

It is not possible to say in what manner the heat affects the body of the child, but it seems that the infant's resistance is lowered and that injuries by food are readily produced. It is not known whether the heat produces changes in the enzymes of the digestive tube, or whether the bacteria make toxic products or irritating acids in the gastro-intestinal tract. It is possible that the epithelium of the gastro-intestinal tract is itself damaged. It would be unscientific to say that bacterially decomposed milk has no causal part in producing summer diarrhea; but on the other hand, proof is insufficient to permit us to say that bacteria play the single rôle. It would seem that the increased heat, particularly of the home, is an important etiologic factor. Whether this be due directly to heat-stroke or whether it be, in the sense of Finkelstein, a diminished tolerance for food and the possibility of injury by food, must remain for the present an open question.

Perhaps this latter point will explain the difference in death-rate between breast-fed and hand-fed infants. The tolerance for breast-milk is always greater than the

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tolerance for cow's milk, and similarly, immunity and resistance is greater in breast-fed than hand-fed children. A word should be said against the practice of keeping sick infants warmly dressed and excessively covered during the hot weather. A cool bath and the out-of-doors temperature are important indications in treatment.

The occurrence of high sick-rate and death-rate during the summer cannot be explained by a single cause. We believe that the condition is due to a combination of causes. Infected milk has a deleterious influence, but it must be considered along with the direct influence of heat, especially the excessive warmth of the home. It would be unwise to deprecate the advantage of sanitary milk, but the problems of hygienic housing, the avoidance of overclothing, the importance of taking the child out of doors and of frequent bathing are important points which cannot be overestimated in consideration of the summer diarrheal diseases of infants.

Finkelstein, after discussing the bacterial theory of summer diarrhea, thinks that infection occurs in those cases in which the gastro-intestinal cells have been previously damaged. He is willing to admit that delicate children who ingest bacteria-laden milk are liable to have gastro-intestinal infection or that damage may be produced by preformed acids and peptones in the milk. The two arguments in favor of infected or decomposed milk as cause of gastro-intestinal disorders were: the increased morbidity and mortality during the summer, and the fact that the greatest proportion of those that sickened and died were bottle-fed infants, while breast-fed children were for the most part immune. But Finkelstein thinks this insufficient. He thinks that the majority of infants who sicken or die during summer time are injured by the heat. The cases begin acutely, show symptoms of hyperpyrexia and the trouble may be cured or even prevented by reduction of the patient's temperature or by providing cool, sanitary

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living conditions. The heat lowers the individual tolerance of the infant and predisposes him to gastro-intestinal disorders which are likely to assume great severity.

If it is asked why breast-fed children enjoy immunity against the injuries produced by excessive heat, it may be answered that the child suffering from digestive disturbances has less resistance to heat than a normal child, since most breast-fed children are well, and a goodly number of hand-fed children are more or less ill, the conclusion is obvious.

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ABSTRACT OF DISCUSSION

Dr. John W. Foss, Phoenix, Ariz.: In Phoenix we have probably the highest dry-bulb temperature and the lowest wet-bulb temperature during the summer, of any place in the world. We have no acute dysentery in children in the summer. We consider the summer the healthiest season for children. In fact, we have very little sickness of any kind among children at that time. So I think this question of heat is practically eliminated as the cause of this class of diseases in children, judging from the experience we have had in our climate. There are few places which have less physical discomfort from heat during the summer than Phoenix. As an illustration of this fact, 15 per cent. of humidity with the thermometer at 106 F. at Phoenix is as comfortable as 70 is at Chicago ordinarily, or the dry-bulb thermometer would have to register 133 at Phoenix to cause as much discomfort as 90 per cent. of humidity with the thermometer registering 98 F. at Chicago. It is probably this remarkable climatic condition which is so conducive to the health of children. Our experience would seem to prove that the mere question of heat has nothing to do with these diseases, especially at Phoenix. The wet-bulb rarely goes above 72 F. in Phoenix in the summer time, while the dry-bulb will sometimes run as high as 110. Evaporation and radiation are very rapid here on account of the pure dry air, moderate wind velocity and varying atmospheric pressure.

Dr. M. A. Weems, Columbia, Tex.: I think this question can be explained by the humidity. In Arizona at the time the air is so dry the evaporation from the body of the child is so rapid that it keeps the body temperature low even in extremely hot weather.

Dr. W. V. C. Francis, Los Angeles: I think one factor in the etiology of summer diarrheas is very obvious, in that the condition occurs in large cities. In Los Angeles we have none of the really poor and none of the overcrowding that you have

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who live in large cities. I believe a large factor in the diarrheas of children is the lack of fresh air. If you remove the slum districts and the overcrowding, you will do away with a lot of the summer diarrheas and deaths.

Dr. Frank P. Gengenbach, Denver: Undoubtedly there are many factors to be considered in the cause of summer diarrheas in children, but I am inclined to believe with Dr. Abt that heat does have something to do with it. Recently we had an early and sudden hot spell for Denver, and in my own private practice I saw distinct disturbances among the babies. This is an instance in which the ideas of Dr. Francis do not apply, for these babies were in well-to-do families, well provided for, well cared for, and had plenty of fresh air. I think that some depressive factor in connection with this hot spell certainly did disturb the babies. Probably in hot weather the babies cannot stand as rich a food, as high in fat, and it disagrees with them. Often it is not necessary to take them off the food, but if I take off the cream and give skim-milk mixtures they rapidly improve.

Dr. Frances M. Allen, San Diego, Cal.: In my experience of six years in Illinois in a children's institution, the children were cared for by mothers who boarded the children for $2.50 or $3.00 a week each, and our results showed that we could keep our mortality low, except during the hot spells. During five years we had a high mortality only during August, during the sixth year, in July.

Dr. L. T. Royster, Norfolk, Va.: To me this is one of the most interesting and difficult phases of the diarrhea question. I come from the Atlantic coast, where the humidity is high, and where a temperature of 88 F. is very uncomfortable, while a temperature of 100 F. out here I have not found at all uncomfortable. In April the temperature rises considerably; then during the first two weeks of May we have a considerably cooler spell, and then the temperature shoots up and the minute the temperature goes up the trouble begins. I can almost predict the number of my calls by the reports of the weather bureau. In June we have a high death-rate. In July and August we have a decreased mortality, so I believe the humidity must have something to do with it. Another interesting fact is this: I have practically no consultations in summer diarrheas from the country. Yet there the temperature is just as great and the humidity is just as great; but I practically never see a child from two miles out with a summer diarrhea. Again, I practically never see a case among breast-fed infants. If a sick child is brought to me in the morning and it is taken for a boat ride for the day, it will come back in the evening 95 per cent. better. In fact, we have tried to train the mothers to take the children to the sanitarium when they first become sick, without waiting to see a physician, even, and it is really surprising how rapidly they improve,

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even though the institution is located but 50 yards from the shore. Another thing is the character of the discharges. These vary from year to year. One year there will be a watery discharge with the same prostration and again the next year we may have a remarkable number of dysenteric discharges, so that from year to year the discharges are markedly different.

Dr. I. A. Abt, Chicago: I think the fact has been brought out by investigators, even as far back as the time of Benjamin Rush, that summer diarrheas are more prevalent in the temperate regions, while further south and even in the tropics, the disease is scarcely known. In my own part of the country there is a striking relationship, I am sure, between the condition of the weather and the summer diarrhea. I think anybody who has lived east of the Mississippi River will not doubt that. Why New Orleans should have a low death-rate with an increase of temperature I confess that at this moment I am unable to see. I am not sure that the heat is the only factor, but it does seem that all over the world where cases are being studied and where men are trying to solve this problem, during July and August there is the highest death-rate. It is true the condition is more common in the cities than in the country, but the condition is not confined to the cities by any means. I brought up this question because it seems to me we ought to study the cause of these summer diarrheas. The war that is being waged all over the world to save babies needs some help from the educator. We urge that every effort be made to save babies and I think it is very important that we attempt to arrive at some definite conclusion as to the cause of trouble. I would suggest that a committee be appointed from the various communities and that this committee be asked to keep a record of their observations and that they submit a report based on such records.

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RICKETS IN ITS EARLY STAGES AND BEST TREATMENT TO PREVENT DEFORMITIES

J. W. COKENOWER, M.D.
DES MOINES, IA.

This subject has not been given much special attention recently, judging from a review of modern medical literature on diseases of children, and especially rickets, but all seemed to have been content with the fact that the disease was due to mal-assimilation with inevitable and unavoidable results and let it go at that.

Rickets is a disease of defective nutrition from mal-assimilation, augmented by the absence of sunshine and fresh air. Some authorities believe rickets due to toxins received by the child through special inheritage, while others contend the toxins are due to alimentation; however, let that be as it may, the disease is essentially a constitutional one and we must treat it as such.

The scrutinizing diagnostician will readily see that the disease is much more common than is generally supposed and will differentiate it by elimination from other diseases in children called something else. There are negatively etiologic factors which must be considered, among which are air, sunshine, hygienic environments, food and age. Like all diseases associated with impaired nutrition, it is less likely to occur among breast-fed children than among those who are deprived of their natural food. For the same reason it is more likely to occur in the latter part of the first year than in the early months, since in so many cases the breast-milk deteriorates in quality after the first six months of lactation and after one year still more so. On the other

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hand, premature weaning, or bottle-feeding, causes chronic indigestion, characterized by excessive acidity throughout the entire alimentary canal which overcharges the blood with acids and disturbs the ossification of the bones.

The disease exists most frequently in the temperate zone and in crowded unhygienic quarters of cities and especially large ones.

In addition to constitutional rachitic disturbances, the bone changes demand early attention, and marked changes are first noticed in the epiphyseals of the long bones most frequently at about the age of 6 months when the organic bone material exceeds the inorganic, and excessive ossification interferes with the growth of the child resulting in permanent hyperostosis.

The age limit is usually between 6 months and 3 years, and the early symptoms are many, among which, before the marked osseous change occurs, are constipation or diarrhea, gastro-intestinal disorders, night sweats, debility, inactivity, often fever, delayed dentition and rapid dental decay, but not typical Hutchinson teeth, nervousness and often convulsions, enlarged abdomen, large head, enlarged and tender epiphyses. All these symptoms may not be present, however, but there will be enough to enable one to make an easy and timely diagnosis, and as the disease is not necessarily a fatal one, it should be recognized sufficiently early to lessen the ravages and shorten the time of the disease so that no bone deformities may occur, and if they do, whether in the early deformity stage or not, there is no justifiable excuse for not preventing the deformity and correcting what exists.

The general treatment in the early stage should be constitutional in its fullest meaning, augmented by dietetic and hygienic measures, prevention of deformities, manipulation, mechanical and operative measures. Medicines are of minor importance compared to food, sunlight and fresh air; however, iron, arsenic, phos-

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phorus, lactophosphate of lime and cod-liver oil may be given with advantage; but since the disease is self-limited, the object should be to tide the child over this period as quickly and safely as possible, remembering that during the softening stage of the bones faulty positions, muscular action, weight-bearing and atmospheric pressure are the main factors to observe in order to prevent deformities; and we should remember Wolf's law of development and equilibrium, that where there is any deviation from the normal perpendicular axis of the body, no matter in what part of the body the deviation or deformity exists, there will be a compensatory one and the entire body must pay the debt, and if this unnatural position be maintained long, the result will be permanent abnormal growth and deformity as in the case of torticollis, in which the bones of the head become flattened transversely, face distorted, eyes deficient and brain undeveloped; few such individuals live to be over 25 without becoming imbeciles.

The same principle is involved in the spine of the rachitic but to a much more serious degree, because nine out of ten such spinal curvatures are upper right dorsal, making convexity on right and concavity on left, causing the ribs to interfere with the normal functions of the organs of the thoracic cavity, especially the heart, which may result in organic lesions and a shortened life. One might thus specialize indefinitely, but generally speaking, rachitis affects the entire organism and produces marked changes in all bony and cartilaginous tissues by means of cell proliferation, and the atrophy is not due to halisteresis, but to the action of the osteoblasts and perforating canals, producing a deficiency of lime in the bones.

Hence, the proper preventive treatment during the stage of the softening of the bones should be to avoid weight-bearing on spine, arms or legs, and child kept on its back; for if it walks or sits, deformities are sure to occur; so the prone and supine recumbent positions

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must be maintained until constitutional and hygienic measures have conquered the disease.

It often occurs that cases are not seen by the physician until the disease has advanced far enough to present one or all of the inevitable deformities; but prompt action by an orthopedic surgeon should overcome such by means of gymnastics, manipulation, mechanical appliances, osteoclasis, osteotomy, tenotomy, myotomy, or tendon, nerve or muscle transplantation.

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RECENT EXPERIENCES IN THE ARTIFICIAL FEEDING OF ONE HUNDRED INFANTS

DURING THE FIRST THREE MONTHS OF LIFE

FRANK C. NEFF, M.D.
KANSAS CITY, MO.

The experiences recorded in this paper were noted during the past year in the care of foundlings in a maternity home for unfortunate girls. No private cases are here included, so that my conclusions as to feeding-results are not as favorable as would be found in the handling of a similar number of cases in homes.[1] The material at hand included infants some of whom were fed at the start on breast-milk, but few of whom continued longer than two weeks. In many of these breast-milk cases the patients were wet-nursed and some fed on mixed milk drawn by the breast-pump. It would be almost impossible to trace any phenomena while the infant was on breast milk to a cause in the milk or wet-nurse, as the handling of the infants was entirely separate from that of the mothers or wet-nurse, about whom I knew nothing except that syphilis had been carefully excluded. Wherever possible an infant at birth or in a nutritional illness was given a start on breast-milk, and this food was, of course, continued as long as breast-milk was available.

All cases were, therefore, problems for artificial feeding at varying periods, beginning at birth or within two or three weeks thereafter.

1. In Arch. Pediat., February, 1911, Dr. Glazebrook describes the Speedwell Country Homes idea of Dr. Chapin, a movement which will be appreciated by all who have had the sad experience of fighting the mortality in a foundling asylum.

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I wish to speak first of the initial loss of weight and the rectal temperature in both breast-fed and bottle-fed infants which our records show.

INITIAL LOSS OF WEIGHT

As has been stated by many observers, notably by Griffith and Gittings,[2] the breast-fed baby continues to lose weight for the first two to four days. The feature in which I was most interested was as to the day on which the child had regained the amount of its birth weight. In Schaeffer's series, 44.5 per cent. of infants were still under weight at the fourteenth day.[3] In the ninety-five cases of Griffith and Gittings, thirty-one did not regain their weight until after the fourteenth day. In my series of sixty breast-fed infants, eight did not show any loss of weight at the end of twenty-four hours, nor later. Four of these were wet-nursed from the start. With the other four, I could find no explanation as to why they so quickly recovered their primary weight. The average number of days was eleven while the longest was eighteen.

CASE 1.—Baby H., weighed 3,725 gm. at birth, had lost 305 gm. at the end of twenty-four hours, began to gain on the fourth day but did not regain birth weight until the eighteenth day, after which the gain was regular. The child showed no febrile nor other disturbance and was nursed by the mother, whose condition was reported good.

In those infants that were given the bottle from the start, and that thrived, the return of the initial weight averaged no slower than the breast-milk babies. One infant on the bottle had about regained its birth weight at the end of twenty-four hours and continued to gain as follows:

CASE 2.—Baby O., weight at birth 3,660 gm., was placed at once on half buttermilk and water. The second day the child regained to within 5 gm. its initial weight, and showed a gain of 100 gm. on the third day. A small amount of malt sugar was then added to the buttermilk mixture, the child continuing

2. Griffith and Gittings: Arch. Pediat., May, 1907.
3. Schaeffer: Arch. f. Gynäk., 1896, p. 2282.

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to gain slowly when at the eleventh day there had been a total increase of 300 gm. above the initial weight.

RECTAL TEMPERATURES IN INFANCY

There is quite a difference in the text-books regarding the normal rectal temperature in the new-born. Rotch states that about a week following birth it is 98.2 F. Fischer places it between 99.4 F. and 100 F. Koplik believes that a temperature above 100.4 F. is abnormal. One frequently hears it stated that the temperature in the mouth is one-half a degree higher than in the axilla, and that of the rectum one-half a degree higher than that in the mouth. Therefore, assuming that the axillary temperature is 98.6 F., it has often been erroneously concluded that the normal rectal temperature is 99.6 F. In several years of routine temperature taking in the children's dispensary, I seldom saw normal temperatures above 99 F. It was for the purpose of convincing myself as to whether 99 to 100 F. is rare and should be regarded as pathologic in infants, that a study of seventy infants was made, first in the infants during the first nineteen days of life, and a second in infants up to six months of age. Sixty of these were on the breast for an average of nineteen days, ten were bottle-fed from the start. Nineteen days was the average that cases were fed on the breast, hence the new-born bottle babies were considered only for nineteen days. No difference was found in the normal temperature of breast- and bottle-fed, and the temperatures averaged the same in infants of this age as in those whose temperatures were taken up to three months, only a few of our cases remaining in the institution up to six months. The temperature was taken twice daily, at 8 a. m. and 6 p. m., just before bathing and feeding. The temperature of one child averaged 97.2 F., while eleven others averaged between 97.5 and 98 F. One infant alone showed an average temperature above 99 F., it being 99.4 F. Only those infants who were not premature, and who were running

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a consistent temperature while they were clinically well, were considered. The average of 2,660 temperatures was 98.6 F. The temperature at 6 p. m. averaged 0.07 F. above the morning temperature. So consistently was there some apparent disturbance in the infant whose temperature ran above 99 F. (only one of the infants in this number had an average reading of over 99 F.), that it has confirmed my earlier convictions that a temperature between 99 F. and 100 F. should be regarded as pathologic.

EFFECT OF PEPTONIZATION OF THE MILK

During the year we frequently peptonized the milk, adding the extract of pancreas and bicarbonate of soda to the bottle ten minutes before use. The milk was peptonized for many of the new-born and for many of those infants who were not gaining in weight, with the hope of finding a favorable effect. An occasional increase had been demonstrated to me by Rosenstern at the Berlin Kinderasyl. For many years in difficult cases physicians have used some form of peptonizing in feeding, often combining it with milk sugar. Undoubtedly we have all seen an occasional good effect. During the past year we have had but two cases where a gain could be attributed to peptonization. One was in an infant fed on buttermilk to which was added malt sugar. There had been no increase in weight for several weeks, the bowels and digestion, however, being clinically normal. One-third of a peptonizing tube was added to each bottle of the buttermilk preparation, with an immediate gain in the infant's weight.

CASE 3.—Baby O., aged 3 months, had gained regularly on malt soup containing two-thirds full milk of 3.4 per cent. fat, but at this time there had been no gain for ten days. The milk was peptonized, the weight increasing 211 gm. in the next two days, and continued to increase thereafter.

No favorable effect in all of forty cases was observed by peptonizing the food of the new-born infant.

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EXPERIENCES WITH BUTTERMILK

The buttermilk which we have used was churned daily in the institution from top milk soured naturally at the room temperature or by the addition of a small amount of the previous day's buttermilk. In warm weather, the top milk frequently becomes sour in half a day at which time it is put on ice until ready for churning. A glass rotary churn has proved satisfactory and can be completely sterilized. As small a quantity as one quart may be churned at one time if desired.

In many of the infants that were difficult to feed, buttermilk was employed, and also in all cases of atrophy. As long as the child thrived it was kept on buttermilk, even through a period of several months, with the gradual addition of top milk. Our experience has been that it exerted a beneficial therapeutic effect on mild enterocolitis cases and that such cases developed less commonly among the infants who had been fed on buttermilk.

CASE 4.—Baby W., a healthy child, weighing 3,530 gm. at birth, fed on the breast for ten days, and then on a sweet-milk formula containing 2.5 per cent. fat for the next twenty days, during which time it gained only 150 gm. At this time we began using half buttermilk and water with the addition of 1 per cent. fat in the form of top milk. During the next twenty-three days the infant gained only 200 gm. The top milk was then withdrawn, the buttermilk was increased to two-thirds, and the water one-third, the infant gaining 800 gm. in the next twenty days, after which it was taken from the hospital.

Buttermilk proved of special value in the summer months, at which season it was better tolerated and produced better stools than the same patient would probably have had if fed on a sweet-milk mixture.

SKIMMED MILK

Some of the infants who had an apparent intolerance for fat and who took buttermilk easily, thrived for a time on skimmed milk. One child that had a simple loss of weight on mixed breast milk showed a remarkable gain when put on skimmed milk with malt sugar. The

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routine was followed with a series of new-born infants of starting them on a dilution of skimmed milk with enough maltose to bring the total sugar up to 7 per cent., as low as one-third skimmed milk to two-thirds water. When an infant ceased to gain or failed to show any increase, top milk was added from time to time to bring the fat content up 0.5 per cent.

This method has given much better results in most cases than empirically starting an infant at birth on a fat-containing milk with a higher caloric content that the infant needs or will tolerate.

MALT SOUP

Thirty infants were fed on malt soup for varying periods. Those that did not thrive were next tried on different quantities of the ingredients.[4] Four formulas were drawn on, named empirically, Malt Soup A, B, C and D.

MALT SOUP A

(Keller's formula, with the exception of the flour, which is here reduced one-half.)

4 tablespoonfuls of malt soup extract, dissolve in 18 ounces of warm water.

4 level tablespoonfuls of wheat flour mixed with 4 ounces of water, and any lumps of flour strained out.

These are added and brought to a boil in double boiler, stirring all the time, and then boiled for five minutes.

Cool and add 11 ounces of whole milk.

MALT SOUP B

2 tablespoonfuls of malt soup dissolved in 18 ounces of warm water.

2 level tablespoonfuls of flour dissolved in 4 ounces of water.

Cool and add 11 ounces of whole milk.

MALT SOUP C

4 tablespoonfuls of malt soup dissolved in 12 ounces of water.

2 tablespoonfuls flour in 4 ounces of water.

Cool and add 17 ounces of whole milk.

MALT SOUP D

2 tablespoonfuls malt soup dissolved in 12 ounces of water.

2 level tablespoonfuls flour in 4 ounces of water.

Cool and add 17 ounces whole milk.

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4. Holt: Diseases of Infants and Children, Ed. 5.

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In these, the quantities of flour, malt soup extract and milk were varied, and were given according to the needs of the individual cases. These changes proved necessary and successful in many cases. Other modifications besides these four regular formulas were also used.

Infants under two months of age did not show the gain found in those who were older. We have used malt soup preparations in the majority of the infants and have been favorably impressed with its tolerance by infants above two months of age. The milk is not to be boiled nor added until the malt soup is cooled, so that there need be no fear of scurvy.[5]

CASEIN MILK

The present German literature regarding experiences[6] with *Eiweiss-Milch*[7] is voluminous and favorable,[8] while comparatively little has been published in this country that has given practical proof of its merits.[9] Those who have seen its use demonstrated by Finkelstein and Meyer have felt that in theory it is correct and works successfully in their hands. That artificial feeding will always be a greater problem in the United States and in those other countries which have such extremes of summer heat than it is in the more moderate climate of Germany is evident. Therefore, it is not to be expected that casein milk will show so favorable results here as reported by the German observers. After twenty-four hours of a hunger diet, the use of a practical whey-free preparation, leaving out the buttermilk, such as we have employed in some cases of alimentary intoxication with diarrhea, was, in the favorable ones attended by an improvement in most symptoms, but the rapid fall in weight was so alarming that the addition of buttermilk or sugar was imperative. When buttermilk was added to the casein milk, as advised by Finkelstein and Meyer, we saw no better results than by the use of buttermilk alone.

5. Thomas: Ohio State Med. Jour., 1907.
6. Welde, E.: Therap. Monatsh., 1911, xxv, 83.
7. v. Reuss: Gesellsch. f. inn. Med. in Wien., January, 1911.
8. Birk, W.: Monatschr. f. Kinderh., 1911, ix, No. 3.
9. Leopold: Arch. Pediat., August, 1910.

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MALT SUGAR

Accepting the German teaching[10] regarding milk sugar[11]—that it is not so well absorbed as maltose, that it frequently causes a rise in temperature, and that it has the smallest influence on the body growth—we have used malt sugar in all cases that did not gain in weight and in many normal cases as a routine. Maltose is undoubtedly slower to ferment in the intestinal tract than is lactose.[12] With the advent of hot weather, it was found that the milk sometimes curdled after the addition of the malt extract, in which case the acidity of the malt was neutralized by the addition of carbonate of potassium, 4.8 gr. to the ounce of glycernized malt extract. Many patients showed a gain in weight which could not be ascribed to any other cause than the change from milk to malt sugar. In some constipated infants, the change to malt sugar markedly improved the condition and frequency of the stools.[13]

CONCLUSIONS

From observations on one hundred infants in this institution during the past year, the following conclusions have been reached:

1. The average length of time before an infant regains its initial weight is eleven days.

2. The rectal temperature of infants under six months of age is 98.6 F. This method of taking should be employed exclusively, so as to avoid the misunderstanding which often exists regarding the indefinite mouth and axillary readings. A temperature of 99 F. or above is suspicious and should be regarded as pathologic.

3. Peptonization of milk showed no advantages in the new-born, but is occasionally beneficial in older infants.

4. Buttermilk is a useful food in some cases, even in the first weeks of life, and where breast milk is not

10. Leopold: Ztschr. f. Kinderh., Dec. 22, 1910.
11. Helmholz: Arch. Pediat., May, 1911.
12. Rotch: Cardinal Principles for Successful Feeding of Infants.
13. Kerley: Arch. Pediat., March, 1908.

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available, should be tried in cases of fat intolerance and in enterocolitis.

5. Some infants showed increased weight when fed on skimmed milk which, when suitably diluted, can be made the basis for fat and sugar additions.

6. Malt soup is the food that in our experience proved the best milk preparation.

7. Casein milk has a useful but limited field in catarrhal enteritis, the alimentary intoxication of Finkelstein.[14]

8. Maltose answers all the requirements for a sugar in infant-feeding.

900 Rialto Building.

ABSTRACT OF DISCUSSION

DR. THOMAS S. SOUTHWORTH, New York: The early regain of weight in such a large number of cases surprises me. In my experience in the hospital the baby's initial loss in weight is considerable. I can confirm what Dr. Neff says as to the peptonization of food for infants. It has largely failed me in my hospital work. Its value, where it is useful, is simply in changing the coagulability of the proteid, but where a child is given a formula which it cannot digest peptonization does not help matters. Malt soup mixtures with suitable milk modifications are very valuable, but it would certainly be a great mistake to go backwards and take one stock formula or one mixture and try to use it for all infants.

DR. M. DRANGA, San Diego, Cal.: I have found that in the case of babies under six weeks old, when they are unable to take other food, I can start them and they gain rapidly on condensed milk, although when admitted they would not retain barley water or any other kind of food. Condensed milk is used only until the babies are able to retain other food.

DR. LANGLEY PORTER, San Francisco: I believe condensed milk has a definite place in the feeding of infants. One of the arguments that is usually made against condensed milk is that it is low in fat and low in proteins. If any one giving condensed milk will study as carefully his formulas as he would in giving any other food, he will find it possible to augment the protein and the fat. I believe the contumely that has been heaped on condensed milk is deserved by the people who prescribe it because they do not use it as carefully as they do other forms of milk.

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14. Finkelstein: Jahrb. f. Kinderh., lxv, lxviii.

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Dr. J. P. Sedgwick, Minneapolis: Regarding the mortality, a word from the northern section may be of interest. The twin cities, Minneapolis and St. Paul, have a population of about 500,000. There are few negroes, little poverty and little crowding. The mortality last year was between 600 and 700. The death-rate is higher during the summer, and although we do have a hot season the heat does not last as long. I remember one August, however, when eighty or ninety infants died. I have found the same condition of which Dr. Royster speaks. During some years we have a preponderance of the catarrhal type of diarrhea and in other years there is a preponderance of the enteritis type. I have been using casein milk and have found it of definite value in certain cases of atrophy and intestinal trouble. In regard to maltose, of which Dr. Neff spoke, I would like to know what form of maltose he used. Some preparations have much more dextrin than others. Maltose itself is rather expensive, costing about $3.00 a pound wholesale.

Dr. R. B. Tubbs, Council Bluffs, Ia.: I cannot see why it is any more legitimate for us to give condensed milk than it is to give proprietary medicines. It has nothing in the world to recommend it but that it is sterilized. Why should we use condensed milk with 25 per cent. of sugar added to it? I cannot understand how a man can say that it does not produce scurvy. In my experience it does not agree with the babies except temporarily. I know the use of condensed milk has brought me more cases in a desperate condition than anything else. As regards the temperature as a causative factor in summer diarrheas, I believe that overfeeding has as much to do with the etiology as anything else.

Dr. A. L. Tilton, Kingman, Ariz.: In the desert country of Arizona we use a great deal of condensed milk. We use it because it is, in a large number of cases, the only thing we can get. Although there are many cattle ranches in Arizona, a cattle ranch is the last place in the world where you will find milk because there is not sufficient grass near the ranch houses for the cattle to be well fed. Hence we have adopted condensed milk as the least evil, and in my limited experience in its use I have seen but one case of scurvy, and in that case no orange juice had been used. I do not think I would have permitted the use of condensed milk but for the fact that it had already been used there with success.

Dr. I. A. Abt, Chicago: There is a great diversity in infant feeding. Professor Keller of Berlin collected formulas of feeding in different parts of the civilized world. He has shown that in England, in Germany, in the United States and in France they have their own methods. The fact is that babies are raised on artificial food all over the world, and in no two countries is the same method employed. I believe that one is justified in feeding a baby according to the best experience

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that prevails in the section where he is practicing. If most babies in the desert of Arizona are saved by feeding condensed milk, then, it seems to me, that method of feeding is justified there at present. We still have much to learn in the matter of adapting artificial food to babies. I am in favor of every man telling his experiences according to the best light he has, and we may, in that way, be enabled to draw conclusions. Answering the gentleman who said it was wrong to use condensed milk because it was a proprietary food, I think it would certainly be wrong to sacrifice the babies for the want of it, if it will save them. I do not wish to be understood as advocating condensed milk, though I should not hesitate to use it if the supply of fresh cow's milk was for any reason unsafe or inadequate.

Dr. L. T. Royster, Norfolk, Va.: I have had no experience in feeding babies on condensed milk. Nine times out of ten the patients brought to me have been on condensed milk, and are in a wretched condition. I am in charge of an institution that takes foundlings. It was formerly customary to feed them temporarily on condensed milk, but I have made the experiment of feeding them on a solution of milk-sugar, and they do just as well. But those that have been put on condensed milk by the physicians have had it given them without any effort to modify it in any way, as many of the physicians knew no more about it than the mothers.

Dr. W. V. C. Francis, Los Angeles: I frequently put children on goat's milk; I believe that is one of the best substitutes for mother's milk.

Dr. F. C. Neff, Kansas City, Mo.: Dr. Southworth asked about the absence of the usual loss in weight of these infants. I did not mean that there was absolutely no loss in weight, but that they had regained it. We simply put them right to the wet-nurse. Whether that had anything to do with their regaining it so promptly, I do not know. As to scurvy, my statement in regard to the boiling of the milk in preparing the malt-soup was simply a quotation from Thomas, who, in 1907, objected to malt-soup because the milk had to be boiled. In my paper I have used quotation marks to show that it was not an expression of my personal opinion. Dr. Sedgwick asked about the maltose we used. The exact analysis of the malt extract preparation we used I do not know.

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THE BABY-TENTS OF CHICAGO

FRANK W. ALLIN, A.M., M.D.
CHICAGO

A study of the last United States Census reports reveals some startling figures. In the whole United States more than 10 per cent. of the infants have died in their first year. This high mortality has not been equally distributed, for grouping thirty-seven of the largest cities, each having a population of over 100,000, the death-rate is about 20 per cent., while in our very largest cities it approaches 25 per cent.—one out of every four. So great is the hazard that no insurance company will issue a policy on this first year. This alarming mortality has aroused many, in and out of our profession, to seek relief.

There was held in Chicago during the summer of 1911, a two-weeks' exhibition, the largest and most comprehensive ever attempted in the interest of child welfare. This idea we copied from New York's exhibit of last year, enlarging and extending in manifold ways to many times the size of that exhibit. This is the culmination of a general interest.

Chicago lost 6,888 babies under one year of age in a single twelve months. A study of the spot maps of the city shows that certain areas produce most of the deaths and far exceed their relative proportion. It also shows the dire effect of the heated season and that certain diseases predominate. After about three days of hot weather, every day of prolonged heat shows a rapid increase in the mortality. Diarrheal cases lead the list, making the total of enteritis jump from below 50 per cent. to above 80 per cent., with a summer average for

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this disease between 72 per cent. and 76 per cent. The bulletin of the Chicago Health Department states that at least 80 per cent. of all the deaths under two years in the summer months are due to preventable diseases.

Many plans have been devised to relieve these first years of their terrible hazard. Infant welfare movements of many kinds are at work and it will be difficult to measure the influence and determine the exact value of each of these. The baby-tent has its place among them. Designed to meet a need not covered in any other way, it has not only met the immediate needs of the individual sick child, but, by example and instruction to the mother, has often taught her to prevent recurrence in this child and her other children. Organized effort to save the baby is relatively a recent thing. Only a few years ago milk commissions were organized, and the idea has spread rapidly until nearly every city now makes some kind of an effort to guard and protect that part of its population living wholly on milk. The mortality was greatly reduced, but remains frightfully high, especially where the population is dense. It became apparent that the milk-supply was not the only thing to be considered. Since the greatest mortality appeared in the crowded districts, it became evident that better air was needed.

The fresh-air wharfs of New York City and the fresh-air sanitariums of Chicago's lake front were among the first efforts to relieve the congestion of our great cities for the little folks. Sending children to the country for outings and tent colonies for certain diseases had proved so successful in demonstrating the benefits of fresh air that fresh air has become one of the important things in the care of babies. Even after the profession had come to see the benefits of fresh air, it was hard to convince the mother. But at present, with almost daily articles in the papers and the efforts of the health department, it is not so much a question of knowledge as of inducing the people to act. Chicago's congested

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districts are at some distance from the lake front. Most of the deaths of babies in the hot summer months occur within quite limited spaces, particularly on the northwest side, in the Ghetto and back of the stock yards. The Lincoln Park Sanitarium and the Jackson Park Sanitarium are caring for many babies, but they are inaccessible to a vast majority in Chicago. Mothers with several small children and a sick baby could not be spared from their homes to go several miles and stay all day at the lake front; consequently the little sufferer remained cooped up at home. To reach these infants, often dying without ever having a doctor or even a fair chance for existence, the baby fresh-air tent idea originated seven years ago.

The baby fresh-air tents are located in the areas where the deaths are most numerous, which is among the foreign population, Poles, Lithuanians, Italians and Russian Jews. The tents are placed either on the roofs of buildings or in vacant spaces in the midst of these thickly populated sections, so near the homes that the mother can bring the sick baby at 8 o'clock in the morning, leave it all day in the care of competent nurses and call for it again at 6 o'clock in the evening. This daily contact of the mother with the doctors and nurses is not the least valuable part of this arrangement. Like the Salvation Army in their work on the street corners and tenement districts, these tents have left the Lake Shore Drive to reach the masses where thev are near their homes.

The equipment of the tents consists of a hospital tent about 12 by 16 feet, with a double roof, board floor and sides, and screen sides above the chair rail. Each contains eight iron beds, a hammock, electric lights, two electric fans and a telephone; also a receiving tent, about the same size, for examining, bathing and temporary care of the baby. A milk station is maintained in this tent where milk in any modification is prepared for the occupants, or sold to the mother at cost. Cer-

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tified milk is used for these preparations. The tents are opened about July 1 and closed about September 15. The tents were kept open nights and Sundays only in extreme emergency of either very sick patients or severely hot weather.

A trained nurse is in charge of each tent, with one or two assistants. There are assigned to the different neighborhoods a visiting nurse and field nurses, who hunt out these sick and poor babies and send or bring them into the tent. An intern is placed at each tent for half or the whole day as the work may justify. He examines all patients, writes the histories, excludes contagious diseases and prepares the patients for presentation to the doctor who visits the tent on that day. The medical staff consists of three physicians for each tent, each assigned to two days of each week. Each is expected to visit the tent at some hour in the forenoon and have complete charge of all new patients entering on his day. Thus each physician has his own patients and prescribes for the others only in emergency. The Pediatric Society has furnished most of the doctors, the remaining places being filled by physicians of the neighborhood having more or less interest in children.

Only patients under about 2 years of age are received, as those older could not be kept in the beds. Only children of the poor are taken. When it can be determined through the visiting nurse of the district that the parents are able to pay, they are referred to their family physician. A large proportion of these little sufferers are brought in or sent in by the district nurse. When patients fail to return they are investigated by the visiting nurse. Not every mother can be induced to leave her baby in the tent at first, but usually after a few days, if her child is not improving as rapidly as it should, she is prevailed on to do so.

About sixty babies under 2 years of age were cared for the first summer, seven years ago. Each year has shown an increase in the number of tents and the number of

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patients, until last year eleven tents were established with a total of 1,750 babies. These tents are located in different parts of the city, no two in the same neighborhood.

The total mortality last year, counting all deaths at the tent, at home or in the hospital within three days after being at the tent, was 1.75 per cent. The year before it was a little over 2 per cent., which was the highest for any year. One of the most successful tents last season gave the following summary:

Total number of patients	363
Average number of times each patient was seen	4
Average number in tent each day	12
Total number of deaths	2
Death-rate for the entire season	0.55 per cent.

One of the grateful mothers remarked the summer a tent was established in her neighborhood: "Last year it was all the time funerals, but now the babies are all getting well."

The baby-tent idea has become so popular that many cities have taken up and followed more or less closely the plan that was originated in Chicago. New York City six years ago put up "shacks," where babies have been cared for in the fresh air. Cincinnati, Cleveland, Minneapolis, Nashville, Rochester and Hartford are some of the other cities caring for infants in fresh-air tents. In Chicago many organizations have become interested and are seeking to do some of this work. Thus far the Elizabeth McCormick fund, through the United Charities, has financed the tents; last year at a cost of a little over $5,000.

The purpose of this paper is to present to the profession a feasible means of getting at one of our great problems of the city, that is, to check the high mortality among infants. The baby-tent idea lays no claim to improving the food-supply, as shown in the milk-commission milk, or to the fresh-air idea, which was in existence long before it appeared. But it has adapted

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these two great ideas in a way and in the place most needed, by scattering tents throughout these congested districts, so accessible that they will meet the need. The number of babies cared for and the low mortality certainly demonstrate the success of the baby-tents and their right to existence and extension to more districts and other cities.

4206 Monroe Street.

ABSTRACT OF DISCUSSION

Dr. J. P. Sedgwick, Minneapolis: There are one or two things I would like to bring before the Section, as they have been of value to us in our work in Minneapolis. We have, running along the ridgepole of the tent, a perforated water pipe connected with the water system of the city, and when it gets too warm in the tent the water is turned on, thus bringing the temperature down. It is not the temperature of the water, but its evaporation that produces the effect, and it certainly does lower the temperature in the tent. The tent has been used four years and the death-rate during the first summer compared with the death-rate in the hospital proper for the same months of the five years preceding was just one-half. The reduction in mortality may not have been wholly due to the tent, but was partly due to the enthusiasm of the nurses and interns.

Dr. May Michels, Chicago: I would like to add that in Chicago after we employed wet-nurses the mortality was greatly reduced.

Dr. Frank W. Allen, Chicago: Instead of the perforated water pipe, on warm days we have had a cake of ice in a tub placed near the fans, so that the air was blown over the ice and through the tent.

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THE NEUROTIC BASIS OF JUVENILE DELINQUENCY

WITH A STUDY OF SOME SPECIAL CASES MOSTLY FROM THE SAN FRANCISCO JUVENILE COURT

PHILIP KING BROWN, M.D.
SAN FRANCISCO

A study of a good many children who have been brought finally to serious account for misdeeds, and especially children who were referred to me from the juvenile court, has shown in almost every case a state of the nervous system in which the symptoms, although often resulting in criminal acts, were none the less expressions of a condition having its beginning long before, in the bad handling of nervous habits, a stimulation of them, or a neglect of them altogether.

It was not punishment that seemed to be needed in a single case, and it is a matter of interest that in the juvenile court alone is crime considered a symptom, as it should be, for which the doctor-judge seeks a remedy, and, if possible, a specific one. A hundred years ago the medical profession emerged from the conception of disease as heaven-sent and from the almost dark ages of a therapy of blood-letting and calomel, and we are now trying to put and keep disease and its treatment on a scientific basis. That crime results only too frequently from a disease of the mind or body, or both, no one knows better than the physician, and it seems our function to emphasize at this time the need for putting the study and treatment of all crime on the same scientific basis.

When the government is sustained by the Supreme Court of the United States in its assertion of its right

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to limit the working hours of women in the interests of the children they may bear, it is certain that forces have been at work on female labor for a long period, making for a deterioration of the race. It is a habit of nations to act all too quickly if honor be assailed, yet when the very life of a nation is threatened by internal conditions, a peculiar apathy generally shows itself.

The awakening to the importance of internal decay has come. The principle of paternalism in republic and monarchy alike has shown a steady extension for several decades. The recent old-age pension law in Great Britain has carried it as far in that direction as it can go, while the effort of France to increase the birth-rate, the almost universal child-labor laws, the educational requirements and government inspection of schools, penalties for the neglect or abuse of children, even to taking them from their parents, municipal nurseries for babies of working women, the special protection of pregnant working women, the prevention of marriage of persons afflicted with a disease dangerous to the consort or offspring, the limiting of the working hours of women in factories, and, finally, the placing of an eight-hour limit in this state (California) on women's work in general, all show the tendency of the government to assume more and more the control of the conditions attending child-bearing, the unborn child and the infant after birth.

It is foolish to prophesy, but a review of the subject during the past twenty years alone indicates a rate of advance within the next twenty years, in the interest of the child of the future, through the study of the degenerating influences of the past, which gives us the hope that a constant lessening of feeble-mindedness, insanity, epilepsy, blindness, mental, moral and physical degeneracy may be brought about through government measures of control.

When Oliver Wendell Holmes said that the education of the child began several generations before its birth,

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he stated a fact that has a constantly widening application. The foundation for the child's nervous system is certainly well under way that far back, and it is in view of this that we must look for and encourage the preventive work of the future, through an extension of government interference, directing the interference by a careful study of the nervous child of to-day, the influences of every possible environment and occupation on him, his home, his parents and associates, work, play, temptation, emotion, excitement—in fact, everything that stirs his feelings in any way. It is with this part of the problem—the nervous child of to-day—that we, as physicians, are brought into contact.

Unfortunately, the problem rarely presents itself seriously to the average parent until it has taken a form far beyond the ability of the family to cope with, or a situation has developed in which the neurotic tendency appears in a form, the importance of which the parents are the last people in the world to suspect, and where some accident uncovers in part a most serious state of affairs unsuspected by the family, frequently with far-reaching ramifications only worked out by patient analysis. Vanity, sulking, unusual craving for notice or affection, irritability, violent outbursts of temper, the unusual persistence of what seems a single bad habit, may be all the parent sees. A few examples will serve to illustrate.

Case 1.—D. S., a very clever girl of 12, through but two grades in five years of school life, nervous and emotional, living with her widowed mother, supposedly a working woman, but in suspiciously comfortable circumstances, is caught, after great effort and weeks of watching, picking pockets in a crowded shop. She was an artist at the job and asserted that she was taught by a man whom she met on the street and who promised her a doll. She said she met him the same day each week and practiced her art in the same shop. Her story was doubted by the police and her mother was suspected of being the accomplice. Examination of the mother showed a bleached blonde in an untidy but showy gown and revealed the fact that amateur prostitution was remunerative far beyond the

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precarious pickings of a child in the weekly shop adventures. She protested her ignorance of the child's acts. The child was congenitally syphilitic and had adenoids and huge tonsils. A little surgery and mercury made a normal child of her, and a chance for some association and memory-tests convinced everyone of her truthfulness. The nervous and emotional conditions attending her promptly disappeared and she promises to be a strong influence in straightening out the wayward mother.

CASE 2.—J. S., a large and well-developed boy of 14, is repeatedly expelled from private school on account of smoking and because of his general indifference. The headmaster of the last school he attended discovered other forms of abuse and, with the father's consent, sent the boy to me. He was a pleasing-mannered boy of the nervous, weak type who had been indulgently brought up in a small town where his father was a leading citizen, influential in the local government. The father had worked hard himself for his position in the business world and hoped to leave his family well provided for. His motives were good and he meant to be a kind and generous father, but he forgot that the boy needed some of the attention the father gave to his business far more than the boy needed the extra dollars. The mother was too nervous to have the strength to do more than worry and had also the idea that children just grew; she had not the faintest idea of the boy's mental processes or physical and mental needs. There was no history of alcoholism, insanity or unusual nervousness in any marked form in the family. Here was merely a weak boy, the product of his environment. The history that he gave me after friendly terms were established was this: He had lied and stolen as far back as he could remember; he had never been shown up with his lying or been caught stealing. Lying frequently saved a long explanation and it seemed always to work. Although he was indulged, it was easier to steal than to ask for money from his parent, so he always stole. It seemed the more certain way of getting what he wanted. For two or three years he had abused himself and had smoked and taken alcohol in some form. He gambled with the other boys in the town where he lived and with them visited houses of ill repute, although claiming only to drink beer while there. He had no power of concentration and his work at school did not interest him at all. He had never taken any systematic exercise, and had always eaten anything and all he wanted. He was unable to go through a day without planning in his mind how he might carry out one of the bad habits to which he had become addicted. His only form of activity, nervous or physical, directed itself in these channels. He had no ambition, no moral sense and no stand-

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ards, and yet this nervous wreck came of a good family and apparently had far more than average advantages and environment. He came up in the juvenile court for stealing a box of apples.

CASE 3.—D. B., the son of a very rich and self-made father, and an exceedingly neurotic but ambitious mother, was dealt with differently by his parents. He was put through the paces by the father, rushed through the lower grades of school, carefully watched and kept at work, all his nervous energy directed into productive and absorbing channels of the parents' choosing, without a counterbalance of play and fun. He had hardly finished grammar school when he was rushed into a business he did not care for, to begin as a slave at the bottom. Fortunately for him, he broke down nervously and physically and after years of begrudgingly-given outdoor life this youth with a forceful and intense nervous activity has regained his physical condition, but has violent headaches from nervous exhaustion and a chronic tic of the facial muscles resembling a bad habit chorea.

The two latter cases represent opposite attitudes of parents, the thoughtless generous indulgence, and the hard narrow grind; they show how harmful two common types of environment may be and how heavily the responsibility of the failures rests on the parents.

CASE 4.—D. T., a girl of 17, whose aunt was epileptic and whose grandmother was alcoholic, locks herself up in a room for twenty-four hours at a time, and finally runs away, because she cannot have her way. She was never punished, or rather treated, for this form of self-indulgence when it began some years before, the excuse being that she was a delicate, nervous child. She ends by hurling anything she can lay her hands on at her mother when she is crossed in her wishes.

Merely ungovernable temper, trained to react the wrong way, and cured only by long continued and wise discipline; what is called in psychotherapy, reeducation. Such a case might easily be the beginning of a dementia præcox, but that unfortunate end could only be hastened by such indulgence and indecision as was brought about by a failure of the parents to understand the conditions.

CASE 5.—Another girl of 17 reads only dime novels, is dull in school, moody, irritable, vain, untruthful, exceedingly dirty in her habits, although belonging to well-to-do and refined but indulgent parents. She finally strikes a servant and the fam-

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ily wakes up to the seriousness of the situation. A properly trained nurse placed in charge of her, away from home, discovers the enormous extent to which self-abuse is carried on. Only the romantic, or possibly the very commonplace, opportunity had been lacking to ruin the girl's life forever. Here again physical, moral and mental reeducation—carried on for years—is the only cure.

An ungoverned temper in a girl of 12, leading her to precipitate a storm if every wish is not granted, is easily traced to the reaction of the apprehensive mother to the child's demands in early youth. The brain storms came easier and easier until the well-established habit held the whole family in terror. The small beginning of such a possible state was shown in my 4-year-old daughter when she soberly announced to the cook that if bread and butter with jam wasn't forthcoming at once, she would "make a dandy fuss." There should be only one outcome to such a crisis, and the parent who neglects the issue or thinks it too "cute" to warrant notice does the child infinite harm.

Babies learn to cry for whatever they want in the earliest days of life. The well-regulated nursery of a maternity hospital is usually as quiet a place as you can find, simply because the routine care leaves the baby always comfortable, and does not allow it to learn that crying brings about some grateful change. Of course if you seek to soothe its crying by any measure out of the ordinary, it goes a long way toward teaching it to cry when it wants that form of relief again. It can be taught to cry in a day of bad handling. Carried further, such treatment teaches the child that a fuss generally brings what it wants, and the training of its nervous system to act in unnatural ways is well established.

Selfishness is an early developed trait, and in nervous children it leads to all forms of trouble, frequently before it is fully appreciated by the parent. A child who cannot have what it wants does not always make a fuss but may sulk, and this may take a form easily escaping notice, yet there may be violent emotional dis-

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turbance in the child. This is far from being the result of self-control, as one might think. The moody and depressed child is a harder problem than the excitable one in most cases, and requires more careful study and training. The ease with which its moods escape detection frequently leads to a form of self-pity and habits of self-indulgence quite innocent in the beginning, but terribly suggestive to the child who finds how much it can do without it being known.

Some years ago I assisted in carrying out a study of the influences of environment on boys from 8 to 15 years old, in which some extraordinary things were shown. A hundred or more boys, all attending school and members of a boys' club in the district where a large number of working people lived, were asked to answer a set of questions bearing on their likes and ambitions. All of these boys had been well trained in playing question and guessing games, and took this matter seriously. The list of questions asked for their favorite boy's and girl's names, favorite poem, song, color, game; what each wished to be when he grew up; what he would do with a hundred dollars if he had it; what he most wanted for Christmas; where he would go if he had a railroad ticket to any place he wished to go in the world; how he would spend a holiday if he had one and could do what he wished.

The result of the study was amazing. A sort of family history of each boy had been made on cards and it included all the names and the occupations of the male members of each family. There was hardly a boy whose ideas carried him beyond the confines of his home and his street. The popular occupation was the father's; family names and the hand-organ tunes overwhelmed all others in their popularity. No shadow of romance could be found in the desire for journeys to African jungles or Arabian gardens. Indeed, the deeply pathetic side of the situation was shown when it was found that dozens of boys in the club had never been to Golden

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Gate Park, the city's great playground, or the Cliff House, reached without change of cars for a five-cent fare, and very few boys had crossed the bay. A very large number had never been north of Market Street, the main thoroughfare dividing the city. In no case did any of the boys want a railroad ticket farther than across the bay. Vigorous manly games appealed to them more than games of chance. The proverbial period of wanting to be president of the United States was not indicated in one boy, nor was there apparent lust for wealth or power, further than that centered in the desire to be a policeman. Two bicycles were the largest things wanted. Altogether, there was not a single unreasonable or incomprehensible answer. I have known many of these boys in the seventeen years that have passed since then, and there are successful lawyers, contractors and business men among them. There are very few who have not moved into better conditions than attended them then.

One cannot but take all this as indicating how sensitive children are to environment, how deeply they are influenced by it, and yet how generally the tendency is toward right interpretations and healthy ideas. I have cited this experience not alone to show the dependence of the child on its environment, but to discuss with you the value to the child of combined personal and class training. We saw the baby's training begun properly in the hospital nursery. We know that if not provided with a harmless and engrossing toy, the creeping infant is quite apt to reach for the live coals of the fire-place or the fringe of the table cover, on which rests the ink and a lighted lamp.

There is energy to be dealt with and, if it be not provided for in a normal and healthy way, the infant is bound to work out its own way which may not be so good. This is equally true of the child, the youth, the young adult, the mature adult and the old man. We count on training and respect for law to keep the

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energies of mankind within reasonable bounds as the age of youth passes, and when we study the transgression of the law we come invariably on the neglect of training, the early misuse of energy, the absence of normal outlets and the continuance of hereditary degeneracy, as one sees so commonly in the alcoholic father and the vicious, highly nervous or feeble-minded child. In inverse proportion to the early training must be the magnitude of the machinery of the law and its forces for order, until finally we come to see that it is the common interest of us all as members of human society, toward which we have a tremendous responsibility, to work for the coming of the utopia in which the interests of the child and the future of the race are paramount to the selfish human interests in tariff on woolen goods, kid-gloves and champagne.

It would be impossible to outline any scheme for the treatment of nervousness in children except to direct the lines along which one may safely go. It can never be too soon to begin, whether it be the child of to-day or his descendant two decades hence.

One who would get into the spirit of the problem should take the time to read two recent books, "The Century of the Child" by Ellen Key, the Swedish leader of the feminist movement in Europe, who has aroused all Germany to active interest in what she calls the greatest problem of the age; and Jane Addams' "Spirit of Youth and the City Streets," in which she gives voice masterfully and pathetically to the cry of the child for a hearing.

SCHEME FOR HISTORY-TAKING IN CASES OF DELINQUENCY, FOR PURPOSES OF PROGNOSIS AND TREATMENT

1. Complaint against the patient.
2. Family history:

   Details of heredity, nationality, social tendency; defects, illnesses and habits; character, occupation, with special reference to contact with poisons.

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3. **Patient's past history:**
   Medical, development, etc.
   Habits, school life, etc.
   Environment and character.
4. **History of present trouble:**
   Sequence of development of nervous habits.
   Sequence of development of bad habits.
   Efforts at correction.
5. **Physical examination:**
   Complete, including eye, ear, nose and throat.
   Evidences of developmental conditions associated with degeneracy.
6. **Psychologic examination:**
   Including analysis of patient's story.

350 Post Street.

ABSTRACT OF DISCUSSION

Dr. John A. Colliver, Los Angeles: For several years I have examined nearly every boy coming before the Los Angeles juvenile court. We have been pioneers in this physical examination as a method of dealing with juvenile delinquents. Nervous irritability has been of special interest to us and we have had many cases. We think environment is the most important cause; 95 per cent. of them are from "broken homes," i. e., homes in which the parents are divorced or separated, or in which one or both parents are dead; or in which the children are left to shift for themselves because of unfit parents. Thus there is absolutely no discipline. So the greatest influence in the production of delinquents is lack of proper parental care, and over this we have little or no control. Criminality begins at home. The earliest symptom is "talking back" without cause, at first with regret, but later with indifference. This is followed by lying; then by truancy from home; later from school; then by bad company; finally by theft, and the boy lands in the court. I believe there is a physical basis for much of this nervous irritability which is the beginning of crime. It is not the natural tendency for a boy to be good any more than it is natural to raise a weedless crop without cultivation. Animals early leave the parent protection and fight for themselves. The deportment of boys, says Hall, is worse at or about puberty than at any other time. The whole organism, and especially the nervous system, is undergoing a rapid change. The boy has to adjust himself to his changing environment. What you could have done with impunity would to-day land him in the court. So the irritability on the one hand and the lack of discipline on the other accounts, I think, for the majority of delinquents. This irritability arises from physical defects, poor nourishment or the like, and by their removal the child gets control of him-

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self and moral improvement follows. We have found great changes following simple operations; while in thirty-six so-called hopeless cases the effects of a needed circumcision was to change the periodicity of their appearance in court from one month to eighteen months and many of them were never returned for misconduct. Reports show them doing well. We have found that boys who are frequently in court often change in moral conduct when nourishment is improved. Again, a boy gets the reputation of being incorrigible when the only trouble is that he does not hear or see properly. We are establishing in the hospital here a department of psychopathy for the observation of certain neurotic children under the assumption that some cases are purely mental.

Dr. L. T. Royster, Norfolk, Va.: The question of delinquency as put forth by Dr. Brown and the question of irregularities in school go hand in hand. I think the causative factors in these cases are physical, environmental and hereditary. There are cases due to imperfect hearing or imperfect vision. The child loses interest because it cannot hear or see and it gradually goes down the line and drops down finally to crime. We cannot get away from environment, and education is a modification of environment; yet back of that, and most important, is heredity. This has been shown in the work of all investigators, from Galton, in England, to Dr. Goddard, in America, who took 2,000 cases of delinquency and put them on a basis of physical and mental factors. In the latter he found three classes, mental delinquincy, imbecility and the "moron," as he terms it. The physical errors can be corrected. Then we have the three grades of mental delinquency. There is one degree that can be corrected; the second class can be taught the ordinary trades. The third is a dangerous class. But in not a single case of these 2,000 children did he fail to find delinquency or feeble-mindedness in one of the four generations back of him. From this come not only the petty thieves but often the drunkards and prostitutes. It has been stated that no woman who enters on a life of prostitution is a normal woman, and I agree with that. Unquestionably genius begets genius, not, perhaps, of the same type, but genius, nevertheless; and the same is true of crime. This may be prevented by controlling these children from the beginning, by proper teachers and finally by the prevention of incompetents from marriage.

Dr. F. R. Branham, San Diego, Cal.: I am tremendously interested in the play-ground movement. But I don't agree with Dr. Colliver when he states that boys are born bad and are going to be bad. They have a lot of vitality. Any good boy that amounts to anything has a lot of it in him and the only thing is to give him a chance to work it off legitimately. The boys in the country seem to have no trouble of that kind, and it is because of the outdoor life. In the cities they do

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not get a fair show. These suggestions of Dr. Brown are all right, but let us look after the normal boy and give him an opportunity to work off some of his steam and we will find that he will get along all right.

Dr. D. S. Hanson, Cleveland, O.: This study of the evolution of the child has been very interesting to me. Some years ago in our country districts the parents were assessed for a few months of school for their children. After a while the teacher was paid by the state, then we came to the graded schools in country districts, and in our cities improvements have gone on rapidly. The child was first furnished free books, then the poor children were given their breakfast, and later they were inspected in reference to their health. In some cities we have a double inspection, one for the school board and one for the health department. The children have their eyes inspected and attention is given to their hearing, and other physical defects noted, and if the same process keeps going on much longer it looks as if in the next forty years the state will take the child from the mother's breast and care for it. Yet I do know that what we are doing for the children is going to have wonderful results. If, for example, every adult had sound teeth, chronic disease would be reduced 50 per cent. in a few months' time. If you improve the children's eyes and hearing, you give them an opportunity to change from dulness and apparent stupidity, oftentimes to wide-awake and interesting children.

Dr. John A. Colliver, Los Angeles: I rise to correct an impression. I did not say that boys were bad. I said it is not a boy's natural tendency to be good. When they are good it speaks well for the parents. The delinquency among the boys in the play-grounds section decreased at once, and I think that is a matter of discipline and training.

Dr. Philip King Brown, San Francisco: I want to state my belief in the goodness of boys and to point out that of course you must give the boy an interesting thing and a helpful thing to play with. He will enjoy it a great deal more. You will accomplish the desired result of directing his energies properly in two ways; you give him something that is positively good and remove the chance of his selecting something positively harmful. Of course we should be connected with the play-grounds association, just as we should with the pure milk commission. We should be connected with the best things, just as we stand for the best things, however indirectly they may seem to bear on the problems of sickness in childhood.

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PYLORIC OBSTRUCTION IN INFANTS WITH MUSCULAR HYPERTROPHY AT THE PYLORUS

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Reports of cases called congenital pyloric stenosis are so frequent in recent medical literature that an apology seems due from any one who would once more bring this subject forward.

Such apology would seem especially necessary when one considers the exhaustive papers of Coutley, Weil and Pehu, Koplik, Morse, Ibrahim and a score of other writers. Yet in spite of the brilliant studies of ten years past, studies based on the consideration of many cases, we to-day, in the presence of one suffering from this malady, are victims of divided counsels. Men of vast experience say: Operate at once or fail in your duty. Men of equally great experience hold operation to be in some degree a lesser form of malpractice. Shall we follow Hutchinson, who finds nineteen-twentieths of his cases, twenty in number, recover if treated medically, or Burghard, Stiles, Scudder and Stillman, who believe that for many of these infants, operation offers the only relief? Perhaps, in the face of such uncertainty, we may be excused for asking once more for consideration of this subject.

We have cared for and studied fourteen such patients and have seen a number of others by the courtesy of colleagues, without opportunity for adequate study. We

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have also made a very exhaustive scrutiny of the literature.

Like all others interested in the matter, we have pondered the different proposed classifications and believe that Nichol offers the most helpful suggestion. He divides his cases in which operation is imperative (major spasm, in his nomenclature) from those in which expectant treatment is indicated (minor spasm).

We believe Koplik's classification, based entirely on consideration of supposed etiology, to be less useful in practice, especially as in it is included the condition of pure spasm without hypertrophy, which we take to be a very distinct clinical entity well proved to arise from hyperchlorhydria, and we have excluded this not infrequent type from consideration here. The question we have to answer is in which of the cases the patients will endure temporizing and which must at once undergo operation. Our own view is that in a large proportion of the cases, obstruction is not absolutely persistent and that in such, dietetic and expectant measures suffice, and also that a few patients who do suffer from persistent positive obstruction, if brought promptly to operation, have a very fair chance to recover. Moreover, operation where successful, offers a complete and almost immediate amelioration of the distressing symptoms, while the abatement of vomiting, wasting and discomfort under medical measures is delayed for weeks, often for months.

Such a division, however, is not always a certain guide; for our fatal cases include two in which the symptoms pointed to an intermittent obstruction; while among our recoveries without operation are at least two instances in which all the clinical evidence seemed to indicate complete persistent obstruction. Moreover, a scrutiny of nearly 300 cases from the literature shows this to be an average experience.

To one of us it has seemed that the type of vomiting had some prognostic value. When the vomiting comes

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on during, or immediately after, every meal and is of the regurgitant type altogether or in part, that is to say, in cases in which cumulative propulsile vomiting is inconstant, one should feel more hopeful for the patient's ultimate recovery without operation; and it would seem that the less dominant the propulsile type of ejection, the more rapid the cessation of symptoms.

A careful search of other observers' reports fails to confirm this view. In very few of the hundreds of reports examined, however, was the type of vomiting reported in detail. We believe a further study may prove of value and we suggest that when such cases are encountered daily notes should be made of the number of ejections, the time of each, the amount ejected, its relation to the ingestion of food, the appearance and the odor of the vomitus; possibly a special chart would prove of value in such investigation. It seems probable that in nearly all the cases the vomiting at first is of the regurgitant type and that, as suggested by Marsh, cumulative propulsile vomiting develops coincident with the beginning of stomach dilatation, so it seems reasonable to suppose that the less severe the lesion the longer the time that will elapse before the appearance of stomach dilatation and propulsile vomiting. A search of the literature reveals that a large proportion of the cases of recovery under expectant measures were characterized by the regurgitant type of vomiting at some time in the course of the disease, usually at a time not remote from the beginning of treatment. This, however, is far from universal, as in the majority of cases of patients reported cured without operation the vomiting is stated to have been propulsile and cumulative, occurring three to six times in twenty-four hours. However, but few of these gave in detail the character of the vomiting from the onset of the disease. Had the cases been studied with this point in mind, we are confident that much valuable information would have been derived, for no individual's experience is likely to be wide enough

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to form the basis of a final conclusion. One point we wish to insist on is that occasional patency of the pylorus, as shown by the appearance now and then of a milk stool, with an increase in the amount of urine and a temporary gain in weight, cannot be relied on as always of good prognostic significance, as witness Cases 8 to 10 of our series, and many reports of other observers. It seems that it may be on the violence of the closure of the pylorus that prognosis depends and it is reasonable to assume that the type of vomiting is to some degree a measure of this violence.

Fritz Talbot has suggested that a good measure of our ability to overcome obstruction at the pylorus may be had by putting small amounts of warm water into the empty stomach. If the water be persistently rejected there is little chance that foods which stimulate pyloric closure will pass into the duodenum. One speaks of violent pyloric closure, for, although we have little but surmise to go on, there are several facts that favor the idea that in these cases we are dealing with a more or less violent spasm of an hypertrophied muscle, a spasm that entirely closes a pyloric way already narrowed by the overgrowth of ring muscle, subcutaneous tissue and mucous membrane. The overgrowth itself seems but rarely to cause complete obstruction, as happened in a case of Morse, which came to post-mortem some months after operation; but this is the only instance of the sort that we could find in the literature. On the other hand, Bloch, Batten and other writers report the pylorus open and the gastro-enterostomy wound closed at autopsies made from six to twelve months after operation. Of course, in considering these cases we must recall to mind that normal growth rapidly widens the pyloric canal while it contributes little to the thickening of its walls. Pfaundler finds 1/10 inch to be the normal thickness of the wall at birth; and this is increased but little by the end of the first year. The lumen, on the other hand, rapidly widens at the end of the fourth week; it is then

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3/10 inch wider than at birth and has gained a further 12/100 inch by the time the child is a year old. When the relation of cross-section to radius is remembered and the fact that the tumor is chiefly a simple overgrowth of circular muscle, itself a constituent of the normal pyloric wall, it will be seen that the normal increase in lumen ought, in time, to neutralize the narrowing due to the encroachment of the overgrown structures of the pyloric wall. This inference is supported by clinical evidence, for if these children can be kept alive until the fifth or sixth month they begin a progressive amelioration and are often exceedingly well-developed babies at the fourteenth or sixteenth month of life.

That in some cases, at least, the condition is one of spasm of an abnormal pyloric muscle already overgrown at the time of birth seems to be reasonably certain. In favor of the antenatal origin of the overgrowth, we have cases reported by Ibrahim, by Dent and by Cunningham, in which the tumors were found in fetal cadavers, and a number of cases in which the symptoms came on at birth, and in which a characteristic pylorus was found post mortem or at operation. Cunningham finds that the folds of mucous membrane, supposed to help obliterate the pylorus in these cases, occur normally; that the longitudinal muscle of the pylorus is proportionally hypertrophied and that many of the individual fibers of the hypertrophied muscles are of the giant type, conditions that obviously could not arise from postnatal hypertrophy due to overwork from closing spasm. And the fact that we have parallel congenital hypertrophies of muscle in the genito-urinary tract and in the intestine (Hirschsprung's disease, for instance); furthermore, that we find the relatively common pylorospasm, due to hyperchlorhydria, occurring in babies in whom one might reasonably expect hypertrophy of the pylorus if it could occur as a result of spasms, and in whom, after weeks or months of such a condition, there is no

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such increase in the circular muscle, is direct evidence that the condition is not of postnatal origin.

But while the evidence does not permit the assumption that the hypertrophy is the result of spasm, the fact of spasm must be accepted, for it is testified to by so many clinical events, as for instance, the intermittence of symptoms, the temporary amelioration that often follows a change of diet and the occasional passage, even in intractable cases, of a milk stool. The degree of the hypertrophy does not govern the degree of obstruction. Some cases with huge tumors have had less intense symptoms than others with relatively small hypertrophy. The expulsive character of the vomiting, too, can only be reconciled with the idea of violent contraction about the pylorus, and the variable intensity of the peristaltic wave suggests that the obstructing force is not of constant measure. Furthermore, Batten's report of a child spontaneously relieved of symptoms, who died eleven months later from a recurrence, but whose pylorus at post-mortem revealed the characteristic hypertrophy, would indicate that spasm played a large part in the production of symptoms in this case at least.

It is easier to find evidence of spasm than to understand the reason for it, and until we do understand the underlying cause our treatment cannot be based on close reasoning and, therefore, must needs be to a degree empirical. We will gain little guidance in the matter of treatment from acceptance of any of the many theories extant, for at the best they are but examples of sapient surmise. Without a doubt, however, study of the laws that concern opening and closure of the normal pylorus, as we can find them in the research of Pawlow, Starling and Cannon, can be relied on to shed some light on the working of the abnormal pyloric apparatus.

For a time it was assumed that hyperchlorhydria was a factor in these cases, but the work of Miller and Wilcox, of Ibrahim, and the findings in some of our cases, negative this view; in fact, whenever trustworthy

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examinations have been made, the secretory power of the stomach, except for rennin, has been normal. Miller and Wilcox found rennin secretion markedly increased in all their cases.

In a recent and very valuable contribution, Cowie has shown that foods of high acid concentration, while in the infant's normal stomach, increase the time of pyloric closure because the duodenum needs a longer period in which to effect neutralization and so initiate the opening reflex. Further, he tells us that foods of extreme alkalinity also tend to lengthen the time of pyloric closure because, as Cannon has shown, there must be a certain mild degree of acidity in the stomach before the pyloric opening reflex is initiated, and the higher the alkalinity, the longer it will take to acquire the needed degree of acidity. It is probable that there is an optimum degree of alkalinity of food for these babies and it seems due them that, whenever possible, we should investigate every such case by methods similar to those used by Cowie working on normal stomachs of babies. We must not overlook the fact, however, that from Holt's service have come reports that lime-water used in moderate quantities increases the production of hydrochloric acid, or that many writers have advocated the use of lime-water in quantities sufficient to prevent rennin coagulation in the stomach and so avoid the stimulus to pyloric closure supposed to arise from the presence of curds in the stomach. Scrutiny of the meals given the spontaneously cured patients here reported, would indicate that the diet plays little part in either the production or the cure of the condition. The course of these cases is practically identical and yet some were fed whey, some condensed milk in varying dilution, some percentage formulæ, and others were breast-fed. It seems irrational to feed vomiting infants food high in fat, yet some writers have reported recoveries while children were being fed mixtures rich in cream.

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Two of the three patients here reported as dying without operation were under observation too short a time for any valid inferences to be drawn from their cases. One died of hemorrhage, a condition several times reported by other observers as the cause of a fatal issue. The second patient died in status epilepticus, the convulsions beginning at a time when lessened vomiting and improving stools seemed to indicate that the child was on the mend. The third died suddenly, within twenty-four hours of the appearance of good milk stools which followed amelioration of the vomiting. This child had an enormous hypertrophy of the pyloric apparatus and yet passed good stools. Such deaths are described in a number of communications and are probably due to the profound metabolic disturbance.

In Cases 11 and 12, in which the patients died after operation, two very young babies were operated on because of alarming rapidity in weight-loss and absolute lack of response to lavage. However, in neither of these cases were these features more marked than in Case 6, in which operation was urged, but was refused, and in which the patient ultimately recovered. This infant, however, had a wet-nurse and was older when it began to develop symptoms.

Patients 13 and 14, recoveries after operation, were the first and second in the experience of one of us, and relief was so instant and positive after operation in both as well as in two other cases, one in Dr. Cheney's practice, one in Dr. Lartigau's (here mentioned by the courtesy of these colleagues), that at that time it seemed that failure to operate in every case would be wrong. Our subsequent experience has shown, however, that in the present state of our knowledge there is little to choose between a course of masterly inactivity and one of masterful intervention. Whether we follow Hutchinson, who decries all operations, or Scudder, who always operates, will depend to a degree on the personal bias of the physician and the problem presented by the individual case.

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By far the best operative statistics are those of Burghard, who eschews gastro-enterostomy and practices pyloroplasty, a point we would commend to the consideration of our surgeons. In spite of Heubner, we believe that stomach lavage with hot, dilute (preferably 1 per cent.) soda bicarbonate solution given once or twice daily, is a measure of great value in the treatment of these cases. External heat applied through the medium of an electric heating-pad or Japanese stove is also of much use. Drip enemas during the time when vomiting is intractable are sometimes, though not always, of use. Condensed milk in moderate dilution is quite as well borne as any other food that we can find for such babies. It is especially of use when combined with mild alkaline laxative.

In conclusion we feel that, while our experience is too limited to allow positive statements, there is evidence to suggest that those patients who retain the regurgitant type of vomiting do better than those whose vomiting is constantly cumulative and propulsile; and we are impressed with the need for a uniform method of studying and reporting these cases which confront us so often and puzzle us so deeply.

2702 California Street—Union Square Building.

ABSTRACT OF DISCUSSION

Dr. W. W. Butterworth, New Orleans: Speaking from an experience of seven cases in the last month does not throw much light on the subject. In one case in which I wanted an operation performed the parents refused consent and the child recovered. This condition occurs more frequently in the first-born and in boys. This patient began vomiting the first week in April and grew steadily worse, and finally was having twenty vomiting spells in twenty-four hours and I thought his time had come. What saved his life was giving him a few drops of belladonna. Then the old negro nurse began giving him a few things and the child began gradually to recover. Dr. Matas had occasion to operate on a child for this condition. The first child in that family had died of the trouble so the parents had some experience with the condition. In this case the tumor was not demonstrable but the character of the vomiting was unmistakable. When one

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feels the hard, fibrous ring there can be no question of the necessity of an operation. But I must confess my inability to say where to draw the line. The best results in my hands have come from the use of stomach-washing, warm bicarbonate solution and the use of antispasmodics. Further than this I have nothing to suggest.

Dr. L. Porter, San Francisco: I did not bring up the question of the clinical picture because there has been so much written on that subject. In common with Dr. Butterworth, I have at times found belladonna useful. Five deaths occurred in these fourteen patients, two under operation. The finding of the tumor was of interest. In many of these cases, in fact, in all of the stomachs I have observed, (not only the pylorus but also the stomach itself is hypertrophied, and the stomach overlaps the pylorus, making it very difficult to palpate the tumor. The interesting question comes up as to why, if these cases have an element of spasm, the tumor does not relax under the anesthetic at the time of the operation.

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A STUDY OF CHILDREN WITH REFERENCE TO ENTEROPTOSIS

RICHARD R. SMITH, M.D.
GRAND RAPIDS, MICH.

If we examine a large number of women with reference to visceral prolapse, we shall find that we may divide them roughly into two groups: first, those who were originally of vigorous build and whose organs were normally or nearly normally placed, but who have given way to child-bearing, excessive hard work or other untoward influences and now present, aside from certain well-marked outward physical signs, more or less sagging of some of their organs; and, second, those who are of the true or essential type, in whom certain fundamental defects in constitution play the chief rôle in causation. For the sake of clearness in this study we shall dismiss the former division, with the brief statement that the prolapse in this class is seldom or never excessive, whereas in the essential or primary form we find it of varying but often of extreme degree. It might be added that the external physical manifestations of the two are very different. The two types often blend and yet the purer forms of each present so many points of difference as to make a clear distinction necessary. I am basing these seemingly dogmatic statements on a study of some four hundred women, a summary of which I presented before another section of this society one year ago.[1] My own interest in this subject has been that of a gynecologist.

1. THE JOURNAL A. M. A., Nov. 26, 1910, p. 1860.

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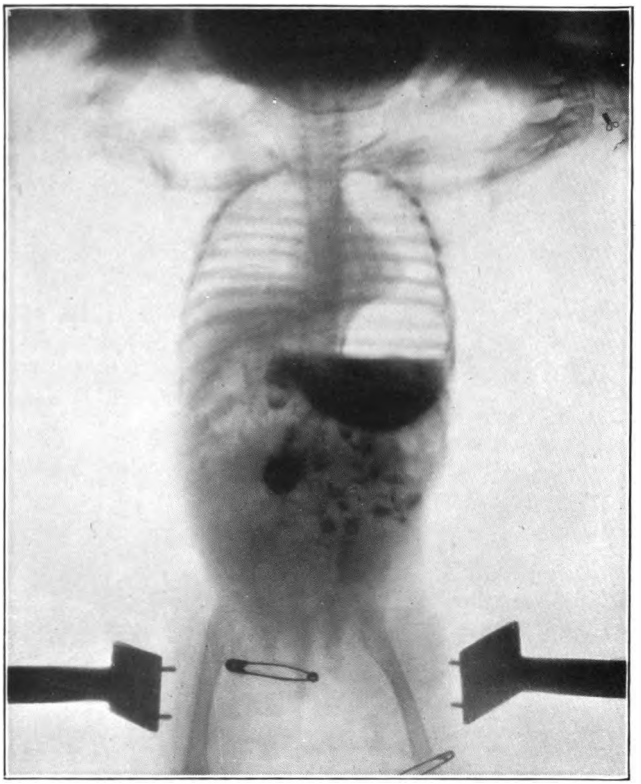

Fig. 1 (B. P.)—Healthy infant, aged 14 months. Note cow-horn shape of stomach, outlined by mixture at the bottom and gas bubbles at the top. Note also its high position, well up toward the diaphragm, the lower border forming a gentle curve nearly on a level with the pylorus. Note colon (gas) in front of stomach, also shape of infant's trunk (spindle- or bobbin-shaped), large in the middle and tapering to either end. Upper abdomen is its roomiest portion.

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The first impression that we get of the enteroptotic woman of the true or essential type, as seen in adult life, is that of frailness, expressed in the contour of her form and often in her features. She is slight and angular and has but little adipose tissue; her muscles are thin and there is almost invariably a laxness and softness of her tissues. These and other structures of the body, including the bones, give one a sense of underdevelopment and a lack of vigor. These may be designated as the funda-

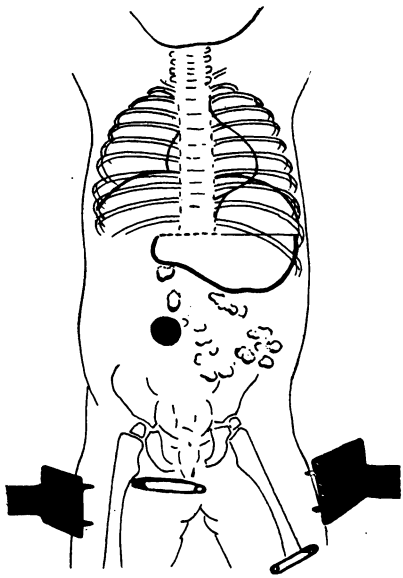

Fig. 2.—A diagram, traced from the radiograph shown in Figure 1 (see Footnote 11).

mental characteristics of the habitus. Associated with and largely dependent on these characteristics, one notes in such an individual that the neck is long, the chest undersized and showing distinct signs of collapse—that is, it is shallow, the upper ribs lie far apart, the lower ones slant downward and inward, lying close together, and there is a distinct lessening of the capacity of the thoracic abdomen. The abdominal walls are soft and lack resistance, and there is a tendency for them to bulge

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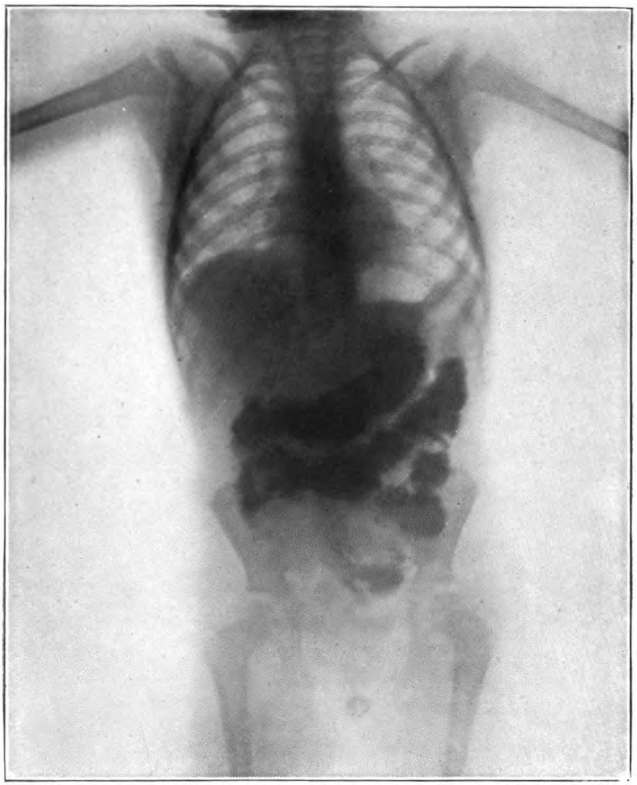

Fig. 3 (M. S.)—Vigorous child, aged 3 years. Exposure twenty-four hours after taking mixture, and mixture repeated before exposure was made. Relation of stomach and colon well shown. Note that the stomach and colon are well up in the abdomen and that both correspond in shape and position to the normal adult organs.

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forward. This bulging is rarely present to any marked extent in the nullipara and in a large number of cases it is practically lacking altogether; in the parous women it is, however, oftener present. The changes in the chest and upper abdomen take place, largely at least, during the growth of the child to maturity. They are permanent changes; at least, they are the most difficult of any material correction. Such women are peculiarly apt to

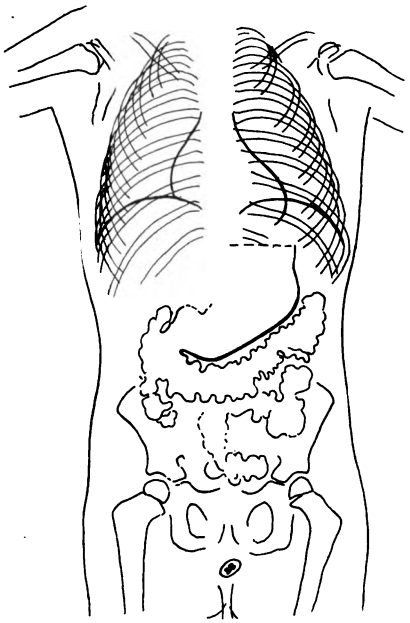

Fig. 4.—A diagram of Fig. 3.

suffer from muscular insufficiency; that is, their muscles are unable to meet the demands on them. It is very apparent in the lessening of the lumbar lordosis, in the rounding back, the forward droop of the shoulders and, not infrequently, in weak foot. The insufficiencies are not always conspicuously present and, generally speaking, are to be looked on as signs of fatigue. They are as a rule less permanent in character and are frequently bettered as the woman improves in health.

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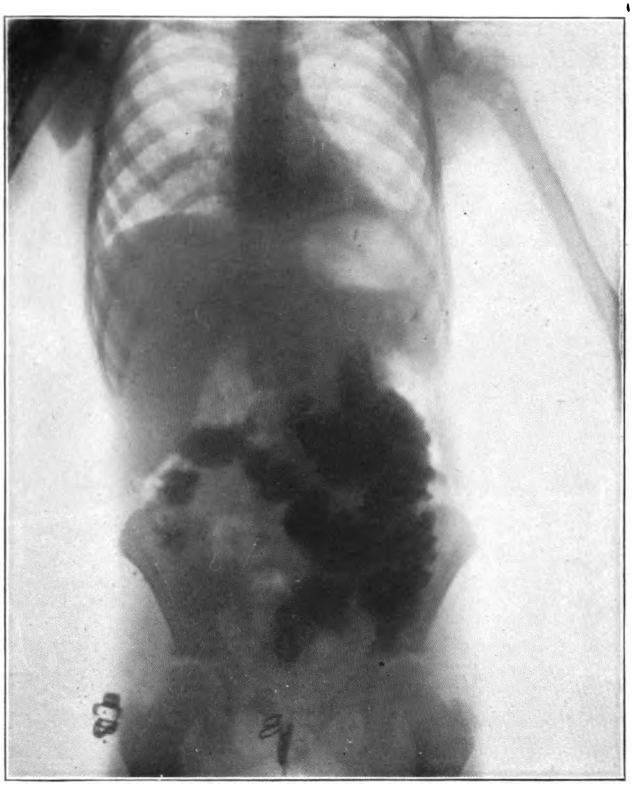

Fig. 5 (Z. G.)—A child, aged 6 years, distinctly of the enteroptotic type. The stomach is high in the abdomen and its lower pole is distinctly hooked. This has a strong resemblance to the enteroptotic stomach of later life, but of mild degree.

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I do not wish to go into the symptomatology more than to say that such women very often have good health in the ordinary meaning of the term, but that they, as a class, have less resistance than more vigorous women and are peculiarly apt to give way to overwork, care and responsibility, child-bearing, an indoor life or poor food. Under such conditions they are brought quickly to a state of fatigue, of lowered nutrition and an abnormal mental condition. They develop pain in the back and groins, a feeling of weight and bearing down in the lower abdomen, disturbances of digestion, menstruation and

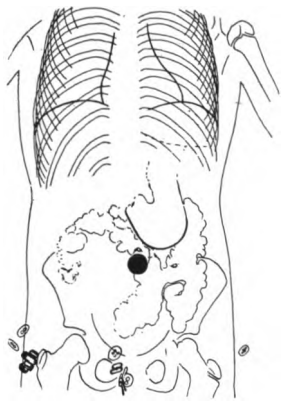

Fig. 6.—A diagram of Fig. 5.

urination and a long series of psychoneurotic disturbances. It may, in fact, be said that the enteroptotic woman, when once her equilibrium is disturbed, develops symptoms that may usually be classified under the term neurasthenia. It is because of the frequency of this occurrence, and the difficulties encountered in seeking to improve such patients, that we are led to make a close study of enteroptosis from all standpoints.

If we inquire into the early history of such women we shall find that, in all the well-marked cases at least, the condition can be traced to early childhood;[2] that is, they

2. That there is here a marked hereditary tendency to assume this habitus may be learned by making inquiry of such patients. I obtained information on this point from some 300 women and found the immediate progenitors to be of similar type in most of the well-marked instances.

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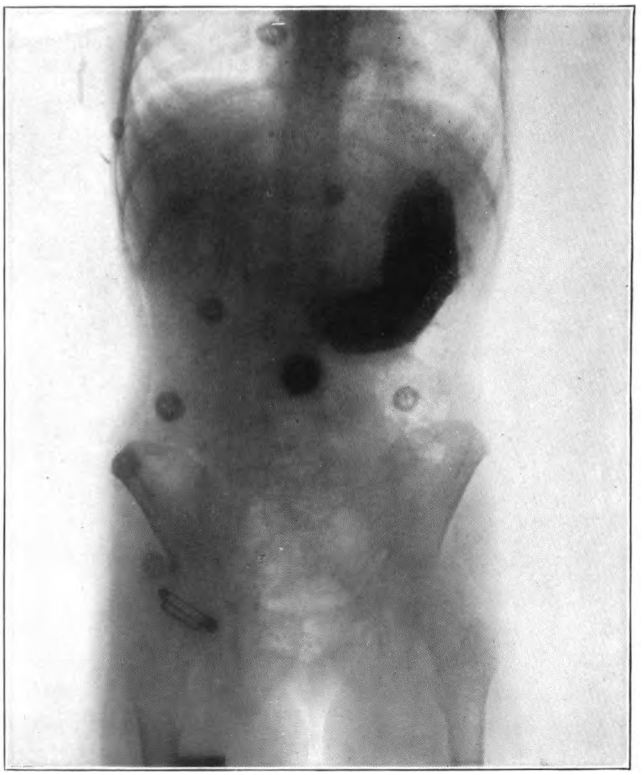

Fig. 7 (A. B.)—Vigorous child, aged 8 years. Stomach almost ideal in form and position.

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will tell you they were thin, frail and perhaps more or less nervous, even at that early period. It was with the idea of learning more of this phase of the problem that I began some time ago the study of children.

W. J. Butler[3] presented before this section at its last meeting a most excellent paper on this subject, based on an examination of 155 children; my findings agree in the main with his.

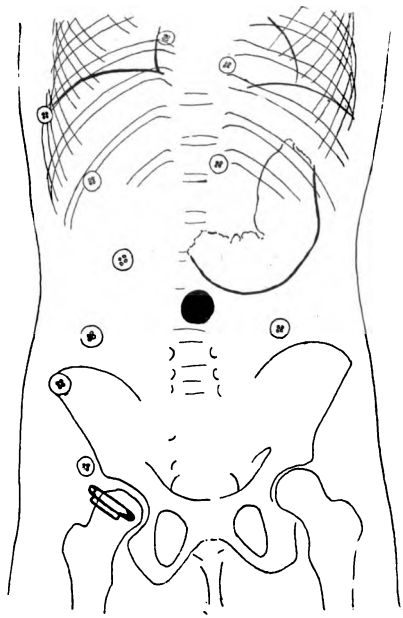

Fig. 8.—A diagram of Fig. 7.

My own study is based on the examination of 109 female children ranging in age from birth to thirteen years. The height was obtained and, following the routine used with adults, the size of the middle zone of the trunk was estimated by obtaining the index of Becher and Lenhoff.[4] After this the general state of nutrition

3. THE JOURNAL A. M. A., Dec. 31, 1910, p. 2279.

4. This index is obtained by dividing the jugulopubic distance by the waist circumference and multiplying the result by 100. This gives, in a general way, the relative capacity of the upper abdomen.

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was observed, the attitude of the child, the size and shape of the thorax (and the epigastric angle). I endeavored to palpate the kidneys and sought the position of the lower border of the stomach under gas[5] and percussion. Some of the examinations were incomplete because of circumstances. Photographs were obtained in a large

EXPLANATION: Each of the following diagrams is made from a radiograph, just as in the case of Figures 2, 4, 6 and 8, but because of the delicacy and even frequently the obscurity of radiographs, rendering them not very clear to one not trained in their interpretation, only the diagrams are presented here. Figures 1, 3, 5 and 7 are presented as examples of the series of radiographs.

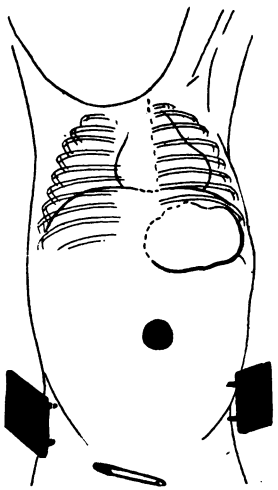

Fig. 9

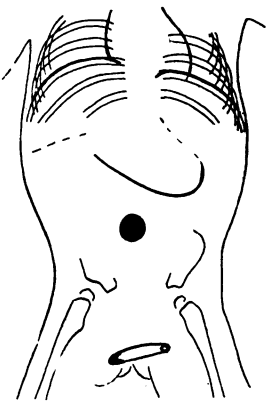

Fig. 10

Fig. 9 (T. H.)—Rather frail infant, aged 8 months. Note high position of stomach; shape not well shown. In original plate, colon, outlined by gas, shows in front of stomach. Incidentally, oblique position of heart is well shown.

Fig. 10 (M. L.)—Frail, poorly nourished infant, aged 14 months. Stomach well up in abdomen. Shape not well shown.

number of instances and x-ray examinations were made in twenty-four cases. Much of this was done to familiarize myself with the peculiar characteristics of the child body at different ages—in this I was much helped by the excellent work of Stratz.[6] No prolonged attempt

5. To the adult a dram of sodium bicarbonate in one-half glass of water is given, followed by one-half dram of tartaric acid in a like amount of water; to the child rather less is given, according to the size and age. Infants received none.

6. Stratz: Der Körper des Kindes, Stuttgart, 1909.

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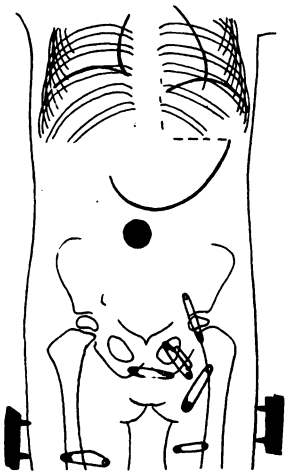

Fig. 11

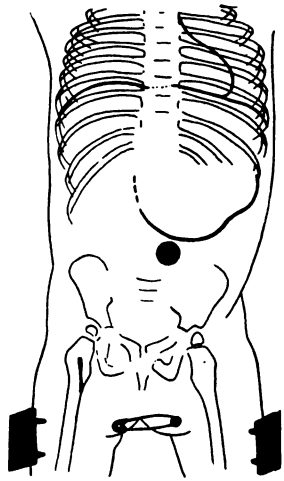

Fig. 12

Fig. 11 (B. H.)—Healthy infant, aged 20 months. Cow-horn-shaped stomach well shown.

Fig. 12 (G.)—Healthy infant, aged 20 months. Stomach rather large. Irregularity of greater curvature, possibly due to peristaltic wave.

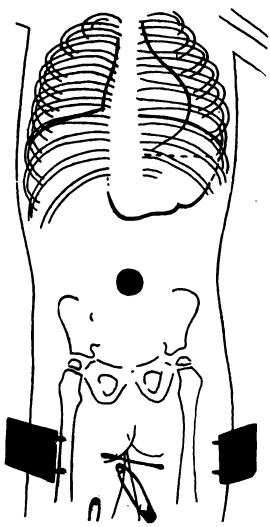

Fig. 13

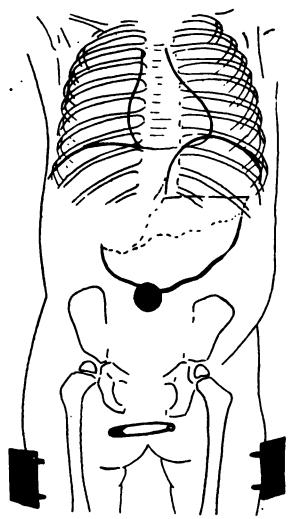

Fig. 14

Fig. 13 (P. H.)—Healthy infant, aged 21 months.

Fig. 14 (K. P.)—Healthy infant, aged 22 months. A large distended stomach; note that it pushes the left side of the diaphragm higher than the right.

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was made to obtain a detailed account of the previous state of health. It was obviously almost impossible to get such from the children themselves and the parents were rarely present. I realize that these examinations might have covered many more points had time and circumstances permitted. The material examined was largely in the D. A. Blodgett Home for Children in Grand Rapids. Through the kindness of Dr. Robert W. Lovett of Boston, I was also able to examine a considerable number of children in various institutions of that city.

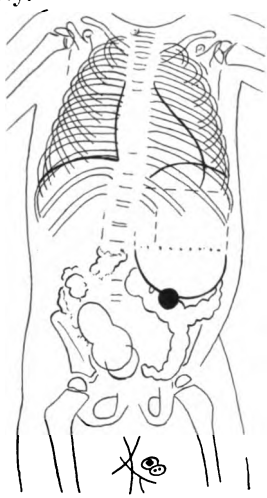

Fig. 15

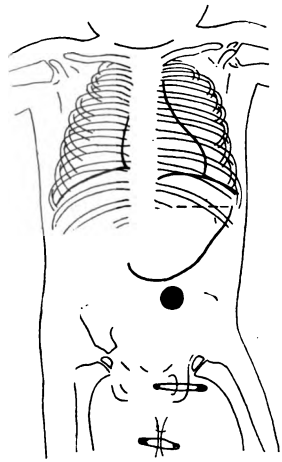

Fig. 16

Fig. 15 (E. W.)—Well nourished, healthy infant, aged 28 months. Stomach rather large, lower border rather low, shape apparently normal.

Fig. 16 (L. E.)—Child well nourished at present, aged 30 months. Shows distinct signs of rickets. Stomach well up in abdomen and normal in shape.

In this series of examinations children of various states of nutrition and vigor were met with, as might be expected—children with slender frames, of little fat and small muscles were common. I was rather surprised at the number of children who presented signs of muscular insufficiency, round shoulders, slight lateral curvature and weak foot. In the thin, frail child this was espe-

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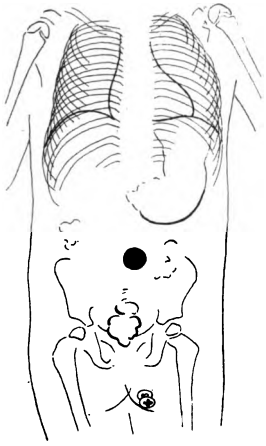

Fig. 17

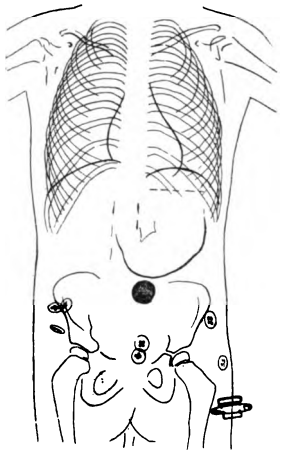

Fig. 18

Fig. 17 (O. C.)—A rather frail child, aged 3 years.

Fig. 18 (E. Z.)—A rather frail child (see Fig. 35), aged 3 years. In this is slightly indicated the hook so common in adult life with the elongated stomach of enteroptosis.

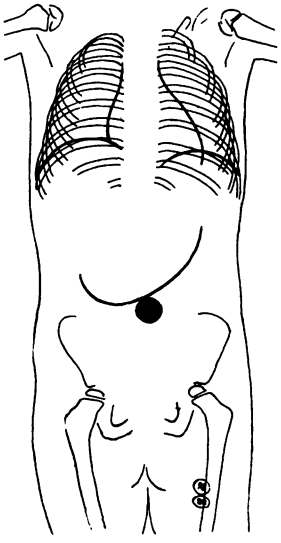

Fig. 19

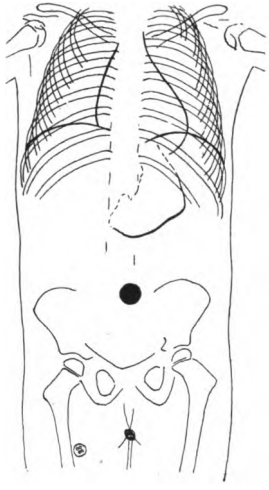

Fig. 20

Fig. 19 (D. V. N.)—Well nourished, vigorous child, aged 3 years. Stomach rather large, lower pole lies rather low.

Fig. 20 (M. P.)—Well nourished and vigorous child, aged 3 years. Stomach imperfectly outlined.

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cially noticeable, though by no means confined to this class. The muscular insufficiency, so frequently accompanying the enteroptotic habit of adult life, is then by no means peculiar to later periods, but is often present early. In order to obtain information as to what constituted normal nutrition in a given instance, it was necessary to have in mind the varying contour of form present at different ages and to compare children of the same age and stage of growth. In the illustrations (Figs. 30 to 37) I have endeavored to bring out the

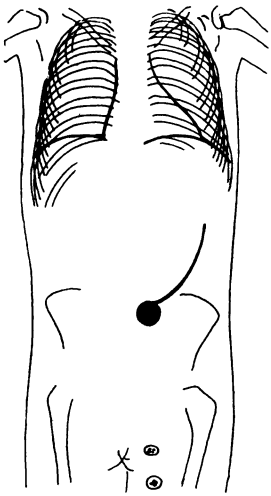

Fig. 21

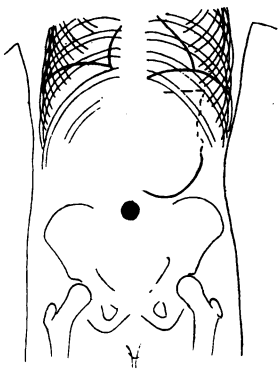

Fig. 22

Fig. 21 (J. V. H.)—Well nourished child, aged 4 years. Stomach imperfectly outlined; lower pole lies rather lower than usual.

Fig. 22 (A. S.)—Well nourished child, aged 7 years (Fig. 30). Lower pole normal in position.

slenderness, lack of fat and muscle, the delicacy of form and feature which characterized the frail child—primary characteristics of the enteroptotic habit of the adult.

The changes in the form of the trunk which normally take place in the growth from infancy to puberty were of much interest, since the capacity of the upper abdomen in adult life is undoubtedly a mechanical factor (although but one) in determining the position of the

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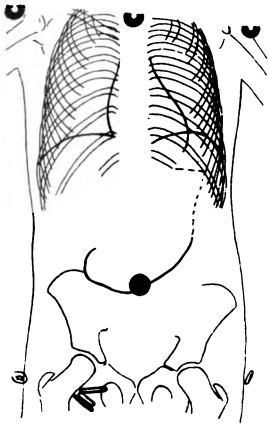

Fig. 23

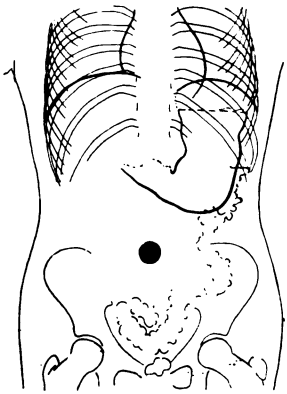

Fig. 24

Fig. 23 (B. W.)—Slender child, aged 8 years. Stomach shows more signs of relaxation than any of the others.

Fig. 24 (B. B.)—Vigorous child, aged 10 years. Stomach ideal in form and position.

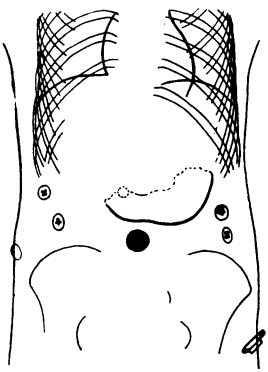

Fig. 25

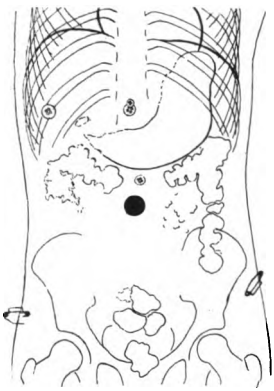

Fig. 26

Fig. 25 (E. R.)—Very frail child (Fig. 36), aged 11 years. Stomach is normal in shape. Some flabbiness of stomach-wall may be conjectured by comparing with Figure 26.

Fig. 26.—Same child as in Figure 25. Picture taken in lying position. Shows lower pole of stomach higher than in standing position.

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contained organs. The shape of the trunk is very different from that seen later on. In the infant and young child it is spindle-shaped, largest at the middle, tapering in either direction to the pelvis below and the neck above. This shape is most outspoken in infancy; as the child grows, the waist circumference diminishes relatively, but this form of trunk remains conspicuous up to the eighth year or the beginning of the bisexual age. At this time begins the widening of the pelvis and the infant trunk-form is soon lost. Not until late puberty, however, is there any marked narrowing of the middle zone. The prominence of the abdomen is most marked in infancy,

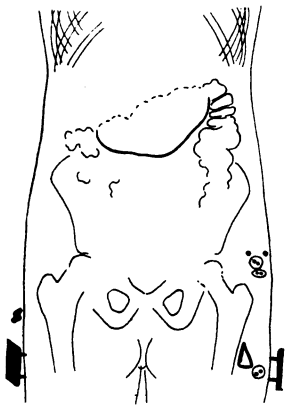

Fig. 27

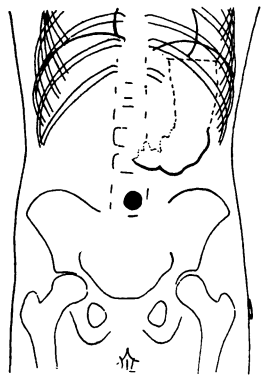

Fig. 28

Fig. 27 (D. S.)—Rather vigorous child, aged 11 years. Stomach well up; colon imperfectly outlined.

Fig. 28 (F. N.)—Frail child (Fig. 37), aged 12 years. Stomach, however, shows no relaxation.

but remains very conspicuous until about the eighth year, when it seems to lessen more rapidly. The shortness of the thorax, the relative depth of it and the upper abdomen, as compared with the adult, are other features worthy of note and are common to all states of nutrition. Not until after the tenth year could distinct signs of actual collapse of the thorax be noted, and this was exceptional then, even in very frail subjects. In a few instances, after approximately the tenth year, it was

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found smaller in size, showing a less vigorous development and some of the collapse so conspicuous in the adult enteroptotic woman could be noted. In most children, however, up to about 12 years, frail or vigorous, there was little change in the actual shape of the thorax and abdomen.

If one has presumed that the marked visceral prolapse, so conspicuous in the frail woman in after-life, is present during childhood, he is destined to meet with a surprise when he comes to investigate the matter. In fifty children, aged from 3 to 12 years inclusive, I sought to palpate the kidneys. The method used was to turn the

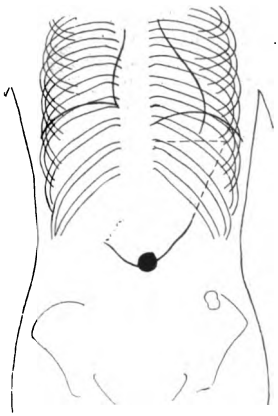

Fig. 29

Fig. 29 (M. S.)—Vigorous child (Fig. 31), aged 12 years.

child well on its side on the table or bed and to reach into the kidney fossa as far as possible with the fingers. With a little patience this was satisfactory. Only such kidneys as could be distinctly felt to slip beneath the fingers were regarded as palpable. The tissues are soft and one can press deeply enough into the side to insure the non-presence of the kidney in an abnormal position. In none of the children (except infants) up to 7 years was the kidney palpable. In one child of 7 the right kidney, and in another of 9, the left was plainly palpable. In a girl of 12 (a very frail subject) the right kidney

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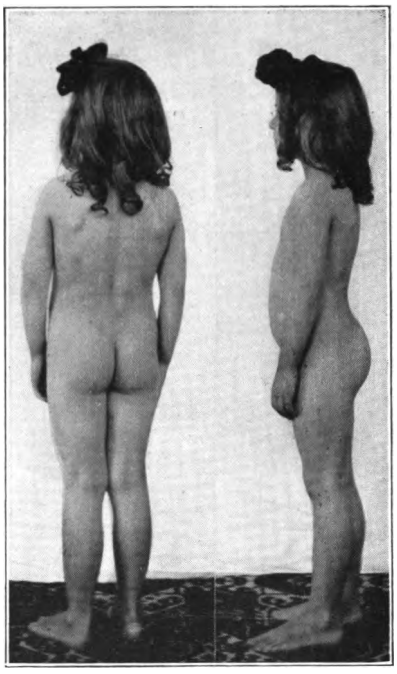

Fig. 30 (A. S.)—Normal child, aged 7, well developed and well nourished. Note contour of form and trunk and erect attitude, which is naturally assumed. Signs of weakness are wanting.

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was palpable. Butler, in fifty girls, found palpable (movable) kidneys present in three, all 12 years or older. This does not necessarily mean that kidneys are never displaced in early childhood, but, when we compare these results with those obtained by examining adult women,[7] the contrast is very striking and we may say that, ordinarily at least, the kidney that was palpable in later life was not so in childhood; in other words, the kidney was not congenitally displaced and remained so. The body habit, I repeat, defined by the fundamental characteristics of frailness, lack of adipose tissue, and slenderness and laxness of tissues, is, however, found very commonly. Of the 109 subjects examined, thirty-nine were infants under 2 years, nineteen under 1 year, and twenty between 1 and 2. In the first group the liver could be plainly palpated in fifteen; the right kidney was palpable in five; the left, in three and both kidneys in two. In the second group the liver was examined in seventeen and plainly palpable in ten; but in the twenty neither kidney was palpable. This, of course, does not mean any congenital displacement of these organs, but, as Butler has pointed out, simply that they can be palpated at this early age because of the shortness of the thorax, the wideness of its lower aperture and the softness of the tissues. After the age of 1 year the palpable kidneys disappeared.

With gas I dilated the stomachs of fifty-seven of these children; this method I thought impracticable in infants and I relied on simple percussion. In all of the children the lower pole was found well up in the abdomen. In fact, in none was there any lowering of this pole which could safely be called prolapse; this was in marked contrast to the examination of adults.[8]

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7. In a series of 100 women, taken at random, I found that the right kidney was palpable in thirty-five and both kidneys in eight. In frail, enteroptotic women the percentage would run much higher.

8. In 100 women examined by this method the lower pole was found below the umbilicus in fifty-two instances, at or above it in forty-eight, Surg., Gynec. and Obst., 1906, iii, 130.

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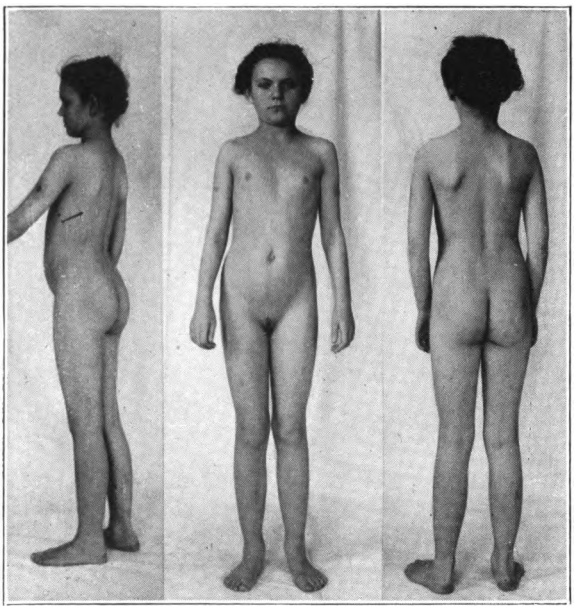

Fig. 31 (N. S.)—Healthy, well nourished, well developed child, aged 12. She stands easily in a normal attitude. Note shape of chest and trunk at this age, also beginning enlargement of pelvis.

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With Dr. Henry Hulst I examined with the *x*-ray the stomach and intestines of twenty-four children. It is to his painstaking and exact work that I am indebted for this unusually fine series of plates. The technic was as follows: The child was placed in the standing position, 5 feet, sometimes 6 feet, from the tube (which was about on a level with the umbilicus) in order to eradicate as far as possible any distortion. The exposures were less than one second in duration. The child stood with its back to the tube, the plate being placed against, but not pressed on, the abdomen. In these examinations a coin was placed over the umbilicus, but I would say in this connection that, in children at least, it forms a very imperfect landmark, since its relation to the spine was not a constant one. The coin might well have been dispensed with. We judged the stomach rather by its relation to the diaphragm, liver and pelvis. Zirconium oxid, mixed with chocolate, sugar and milk, we found a fairly palatable mixture and free from any nauseating or ill affects, a thing which cannot be said of bismuth. The child was given 8 ounces to a pint of the mixture, at noon on an empty stomach and the exposure made immediately afterward. In twenty-four hours another similar meal was given and a second exposure made. This was done, of course, to outline the stomach and colon together. The results were as follows: The stomachs, as outlined by the mixture, varied in size and shape. As contrasted with adults, the stomachs were well up in the abdomen and the hook at the lower pole, so common in the enteroptotic stomach of the adult, was seen in but few instances and then only in comparatively slight degree. The amount of gas in the stomach and intestines seemed to influence the size considerably, this being often changed at the second examination. This seemed to have little or no relation to the vigor of the child. We would naturally turn to the frail child to find the visceral prolapse which we seek, but even here, except in one instance (Fig. 23), it was but slightly

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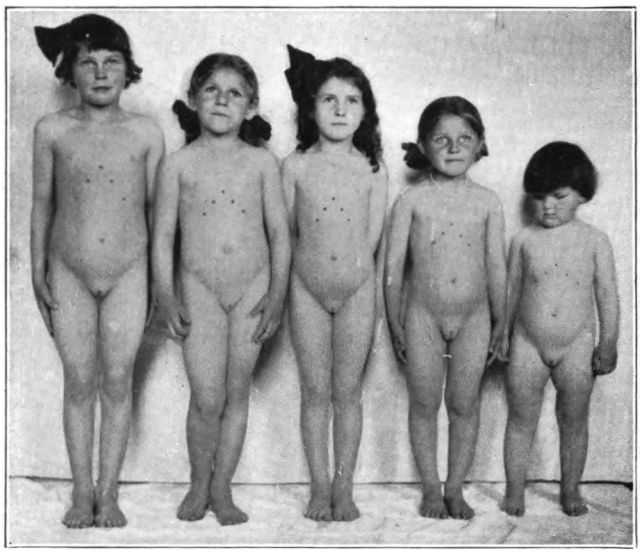

Fig. 32.—From right to left: M. Z., E. W., J. M., A. S., P. C., aged 2, 6, 6, 7 and 8. The middle one is slightly frailer than the rest, but all are of vigorous type. Note amount of fat, good development and the few signs of weakness. Note also the wide epigastric angle, which is a constant one in childhood in all types. Some narrowing is occasionally found after 10 years of age in frail children, but this is not the rule. Narrowing is the rule in enteroptotic women and is an accompaniment of the underdeveloped, collapsed thorax.

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indicated as compared with the frequently found outspoken abnormalities of later life.

The adult stomach in position and shape stands in a more constant relation, I believe, to body habit than the kidneys and, therefore, merits our study. What shall we regard as the normal stomach? I believe with Hulst[9] and Holznecht[10] that the small cow-horn-shaped stomach, with its lower border about on a level with the pylorus, must be regarded as such. In this connection we must dismiss from our minds the matter of function and regard only the anatomy, for the two stand in no constant relation. It not infrequently happens that stomachs that are elongated, even to extreme degree, will care for food in apparently a normal way. If we disregard function and think only of the anatomy we shall be arguing to the same point. Personally, I rather like the term "ideal stomach" to designate the small cow-horn-shaped stomach just mentioned. It is mechanically correct and is the form from which all others seem to deviate. As Holznecht has pointed out, this is the only constant form of stomach found, the more frequent form with the greater curvature dipping downward in a hook varying widely in the degree of this formation and forming a series of gradations, some of which approach the "normal." He points out also that this is the stomach that should perform its functions most easily; an engineer would build it so. It might be added that the "ideal stomach" is found only in vigorous individuals (adults) and that the elongation found in others varies much according to the degree of frailness and relaxation which they present. If now we add the fact that the "ideal stomach" is the common one of infancy and childhood (or, in other words, that the elongated, stretched-out stomach of later life was originally of this form) we have another argument for the establishment of this conception.

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9. Hulst, H.: Physiol. and Surg., Ann Arbor, Mich., September, 1905, p. 391; Arch. Phys. Ther., January, 1906.
10. Holznecht, G.: Berl. klin. Wchnschr., Jan. 29, 1906, p. 127.

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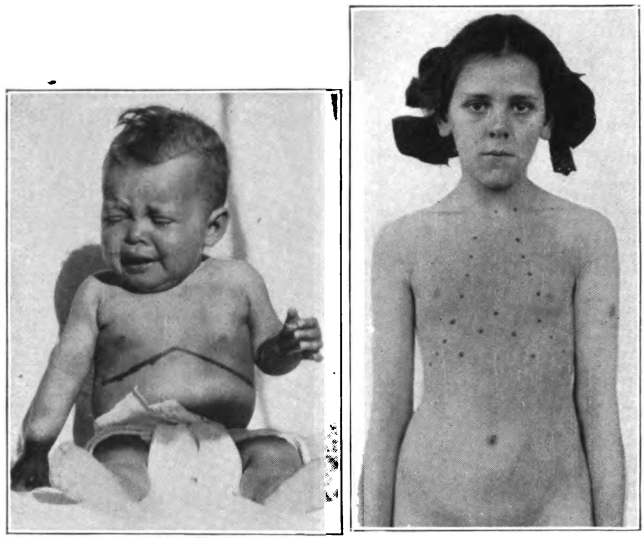

Fig. 33 (M. S. and M.)—Shows difference in shape of trunk in infancy (16 months) and at 12 years. Note also the length of thorax and epigastric angle in the two.

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The results of these x-ray examinations may be best seen by examining the reproductions of the plates.[11] The youngest child examined was 8 months old, the oldest 12 years. In some of the younger infants the mixture seems to have escaped quickly into the small intestine. In one or two the transverse colon, as outlined by its contained gas, lies apparently in front of the stomach. We found this in infants, but never in older children. In the younger infants the type of stomach prevailing was of the cow-horn-shape. Not until the age of 3 years did we find evidence of the hook at the lower pole, and then only in slight degree. During the whole of childhood, although the stomach was sometimes large, it was found, comparatively speaking, well up in the abdomen, the lower pole showing but little tendency to hook. (Figure 23 forms an exception to this.) Changes in stomach-form in successive pictures were sometimes seen, especially in the older children, but only when well indicated were they considered as demonstrating some relaxation of the wall. In the adult the more nearly the ideal the less can such changes be demonstrated. This applies to the changes brought about by shifting of body position as well as those made by varying amounts of stomach contents.

We may conclude a number of things from this study. The enteroptotic habit of adult life finds its counterpart in the frail child presenting the same fundamental characteristics: Frailness, lack of fat, slenderness of muscle, lack of vigor in body development. Any actual collapse of the thorax or diminution in the capacity of the upper abdomen is seen only in older children, and then only exceptionally, even in frail subjects. The muscular insufficiencies of later life in enteroptotic women are

11. It would have been desirable to make direct copies, but these, in the ordinary half-tones, are so blotched and indistinct that drawings were substituted. Four of the plates are, however, offered. The drawings were made according to careful measurements, directly from the plates and give in a true manner the relationship we seek. In most instances but one of each child is given. The position of the colon is reflected quite closely in that of the stomach.

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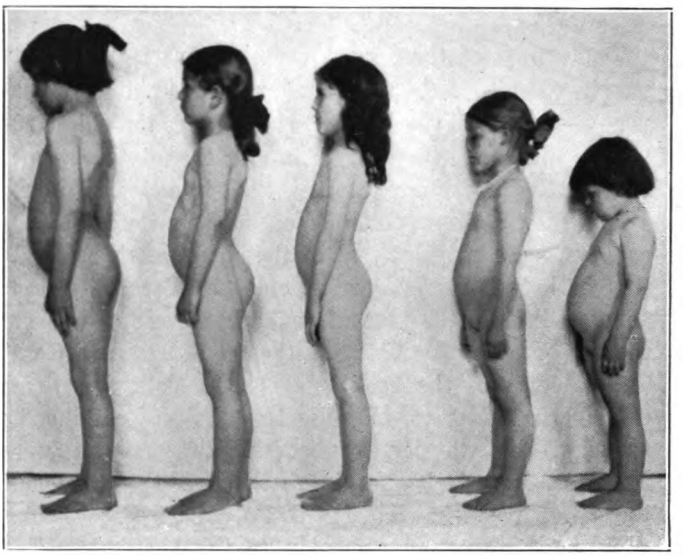

Fig. 34.—Same group as in Figure 32. Shows the prominent abdomen found in childhood. These are all vigorous children.

Fig. 35 (E. Z. and M. S.)—Both children aged 3. The one of vigorous, the other of frail type. Note slenderness of form and lack of fat in the one.

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common in frail children. The prolapse of kidneys, stomach, colon and intestines which accompanies the enteroptotic habit of adult life is not outspoken in childhood (under 12 years) except in rather rare instances, and then usually late in that period.

In my opinion, the habit itself is of far greater importance to the enteroptotic woman than the prolapse of the viscera which accompanies it; at least, in the vast majority of instances. Her lessened ability to fulfil the duties imposed on her, the limitations placed on her by her lack of vigor and the unhappiness which such women in a state of fatigue endure are serious matters with her. I believe that more might be done in the way of prevention. The frail child, it seems to me, should be more clearly recognized as an entity and its tendencies more intelligently fought. In a large number of instances, at least, I believe it possible to maintain a fair state of nutrition in such children and to direct them to a more vigorous womanhood. When such is uniformly done, we shall have accomplished much and shall do away, to a certain extent, with the enteroptotic, neurotic women, who form such a problem in every-day practice and in almost every specialty.

ABSTRACT OF DISCUSSION

Dr. William J. Butler, Chicago: I do not suppose any one in this country has done more than Dr. Smith to call our attention to enteroptosis and to put it on such a basis as to make us realize just what it means. Enteroptosis in the adult may be divided into two types, the congenital type and the acquired type. The acquired type is never seen in children, and is the result of the laxity of the tissues, a muscular insufficiency. The congenital type is quite commonly found in children, in fact, one often wonders whether it may be regarded as an abnormal type. In referring to types I refer to the body form of the individual. In children there is no doubt we can see right from the earliest period, examples of the enteroptotic habit, i. e., the body form. It does not show itself frequently, however, until the child begins to grow and develop. In discussing enteroptosis we must keep in mind not merely the matter of organ displacement. The organ displacements are merely the consequences of this body type. As a matter of fact, what would be

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Fig. 36 (A. B. and E. R.)—Two children, aged 11. Contrast contour of form, amount of fat, muscular development, bony frame and size of chest.

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enteroptosis in later life is a condition that is constantly present in infants, i. e., so far as the palpability of the liver and kidneys is concerned. The stomach, however, is not below the umbilicus. The enteroptotic body form of the child begins to show itself quite distinctly just before puberty, from the tenth year onward; i. e., the characteristic frailty of structure and the lack of tone. The organ displacements, however, are not to be found, except in exceptional cases, much before the twelfth year. I noticed that in Dr. Smith's cases, as in some of my own, the kidneys become palpable about the twelfth year. They may be found exceptionally about the tenth year. This is likewise true in stomach displacement. I did not find any below the umbilicus until about the twelfth or fourteenth year; then occasionally I found one. A thing that particularly strikes the gynecologist is that this matter of enteroptosis is one of the most serious problems that can possibly be considered because of the innumerable cases of ailing women in whom there is an enteroptotic habit which had its origin in early childhood and which is responsible for all their symptoms. In some of these patients it is surprising how long they can carry the organ displacement without symptoms; and in others it is equally surprising how marked the symptoms sometimes are with but slight displacements. The condition should be recognized early, and it is important that they should always be kept above par from the nutritional standpoint. This should help to prevent the development of their symptoms, which are of a neurasthenic character. These individuals are always of the neurasthenic type. In fact, it is sometimes a difficult question to decide what is the cause of their symptoms, the nervous instability or the enteroptotic habit, as they are associate conditions. The symptoms of which these patients complain do not occur until some other condition precipitates them, e. g., child-bearing, or a serious illness, or any prolonged physical or nervous strain. The prophylactic step is to keep these individuals in a high state of nutrition at all times.

Dr. I. A. Abt, Chicago: Several years ago I read a paper on "Floating Kidneys in Children" at the Atlantic City meeting. One of the cases I described at that time was a little girl from the Orphans' Home. The kidney was excessively movable, descending down into the pelvis. Sometimes the ureter became twisted on itself and she was seized with violent colic. We applied a pad which she wore for a long time and she was relieved of discomfort. I have followed up this case. She is now 20 years old and I had opportunity to examine her recently and found that the kidney was no longer mobile and could not be palpated. I would suggest, therefore, that possibly some of these patients may recover

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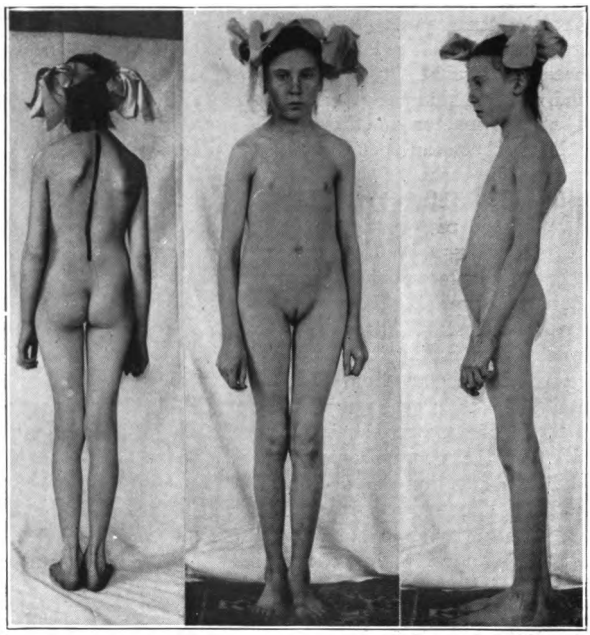

Fig. 37 (F. N.)—Frail child, aged 12, showing muscular insufficiencies and slight lateral scoliosis. Chest remains distinctly behind in development.

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spontaneously. Another point to which I would refer is that we are liable to fall into error about the size of the child's stomach. Not long ago, I saw a child at a hospital which had the symptoms of pyloric stenosis, though no tumor was felt, i. e., there was the characteristic vomiting and peristaltic wave. The case came to operation. On opening the abdomen, it was found that the stomach, after being emptied, was not nearly so large as had been thought. Therefore, I believe that in estimating the size of an infant's stomach, the organ should be examined after its contents have been evacuated as well as in the distended condition.

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REPORT OF BRAIN CASES

WILLIAM J. BUTLER, A.M., M.D.
CHICAGO

During the past year a number of interesting cases involving the nervous system occurred in the service of my colleagues and myself at the Cook County Hospital, a few of which I herewith report.

CASE 1.—Johanna W., aged 8, of German parentage, but born in France. She was brought to the United States when 3 years old. No serious illness had occurred during the first few years of life. At the age of 6 she started to school, which she had to give up shortly thereafter on account of headaches. At that time the father noticed some change in the right eye and later a similar change in the left eye. The affection was of such a nature that she could see better in a dim light. She was treated with but little success for several months. The sight thereafter gradually improved and her headaches were relieved to the extent that she was again able to attend school. About a year ago the headaches returned and were very severe. Sleep was much disturbed and the only comfortable position when in bed was with the head lower than the trunk. She had not vomited before entering the hospital. Several months ago she had measles. This seemed to aggravate her condition. It was noticed about this time that she used her arms awkwardly, stumbled in walking, and had difficulty in talking. While out walking on one occasion she stumbled and fell. The injury did not seem severe, but immediately following the fall she had a slight convulsion which was not accompanied by unconsciousness. Her symptoms gradually grew worse. Walking became more difficult, her speech less distinct and she seemed to have some trouble in swallowing. She was brought to the County Hospital October 3, 1910, and walked into the examining room.

Family History.—Father and mother healthy. No history of syphilis. This was the only child.

Examination.—The patient was a girl of average height for her age, but undernourished; no tenderness of head nor rigidity of neck; face rather expressionless, mouth open; throat negative; no cervical adenitis; chest symmetrical and well formed.

Lungs: Percussion negative, auscultation negative.

Heart: Area not increased, tones normal, no murmurs.

Abdomen: Liver and spleen not enlarged. Abdomen otherwise negative.

Extremities: No edema or eruption.

Nervous System: Mentally clear, but unable to speak distinctly. Later she was able to answer questions only by nods and shakes of the head.

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Muscular System (motor power): No absolute paralysis, but paresis. The left abducens was paretic; slight horizontal nystagmus.

Face: She moved only the left side of the face and that but little; could not protrude tongue nor corrugate lips; opens mouth; palatal reflex present but patient could not be induced to raise palate voluntarily.

Arms: Marked spastic paresis of left arm which was held in extension; slight spastic paresis of right arm.

Legs: Held in strong extension; partial flexion was possible but was never maintained; marked spasticity; the left was much more involved than the right.

Trunk: When placed in sitting posture patient had difficulty in maintaining her position. The spine showed scoliosis which was easily corrected.

Reflexes: Arm-jerks active, knee-jerks exaggerated. Babinski present on both sides. Oppenheim present. Gordon not elicited. No ankle or patellar clonus obtained.

Sensation: Touch intact; pain, analgesia of left side; temperature, intact; muscle sense intact; coordinated movements not well performed; patient had to be fed, but the difficulty seemed to arise from stiffness and weakness primarily. Taste and hearing so far as tested were all right.

Examination of left eye showed a retinitis.

Spinal puncture was made several days after admission to hospital. Pressure not increased; fluid clear. Prolonged centrifuging revealed very few polynuclear cells and lymphocytes and no bacteria.

Pirquet skin reaction was made and found negative.

Wassermann examination of blood was negative. No leukocytosis. Urinalysis was negative.

Subsequent Course.—In the subsequent course of the disease all symptoms became more pronounced and the patient passed into a state of profound coma, which continued for a few weeks before death, which occurred Dec. 27, 1910. She had in this period, several times a day, a slight general convulsion, during which the whole body would become rigid and the face flushed. Nasal feeding was necessary for several weeks. Decubitus developed on dependent parts of the body. The pulse was accelerated during her stay in the hospital, and she had at times a rise in temperature. The respiratory rhythm was disturbed during the period of coma.

Diagnosis.—In view of the general symptoms of cerebral pressure, namely headaches, retinitis, later vomiting and increasing unconsciousness, and in view of the focal symptoms, especially the paralysis of the right facial and the marked spastic paresis of the left arm and leg, in contrast to the slighter paresis of the right arm and leg and left facial, also the dysarthria and dysphagia, the diagnosis of a brain tumor located in the pons was made. In investigating the etiology of the tumor, it was found that the blood was negative to the lues reaction, and that she gave a negative skin tuberculin reaction. While this latter point could not be decisive against tuberculosis, a negative reaction might be of greater value in a diagnostic sense than a positive reaction. Consequently it was thought that the tumor was a glioma, or might prove to be a solitary tubercle.

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Necropsy.—The postmortem was made by Dr. Boughton, whose diagnosis was as follows: 1. Glioma of pons. 2. Decubitus on both trochanters, right ilium, sacrum and external maleolus. 3. Parenchymatous degeneration and slight passive congestion of the liver. 4. Fatty degeneration of the kidneys.

It will be noted from the history of this case that fully two years before death symptoms of brain tumor started. The process had evidently existed for some time before distinctive localizing symptoms developed themselves. The importance of recognizing brain tumors early is self evident. If we can satisfy ourselves that they are not luetic, and to this end I would not depend on a negative Wassermann but also resort to antiluetic treatment, there is only one course of treatment open, and that is operation, first for the symptomatic relief, and second, if possible, for the removal of the growth.

CASE 2.—Wanda M., aged 8½ years, admitted to the hospital Nov. 16, 1910; of German parentage but born in South Africa. Three weeks before admission the child began to feel somewhat indisposed, did not care to go to school, lost her appetite, complained of headache, was feverish and vomited twice or three times. The mother insisted on the child continuing in school for about three days following her first complaints. The headache grew worse and she was taken from school, with all symptoms continuing and exaggerated. In the course of a few days she began to tremble in the legs and arms. This was mild in character until about five days before entering the hospital, when the child began to tremble all over. She complained of stiff neck and back and said she had pain everywhere, but especially in the head, shoulders and arms.

Previous History.—Had had measles and small-pox.

Family History.—Negative. There was one other child who was healthy.

Examination.—The patient was a fairly well nourished girl of 8 years; the skin was pale and her expression was anxious. She breathed with the mouth partially open. She seemed to be in great pain, was restless and irritable. Every few minutes the pain was so great that she went into a semi-clonic condition all over the body and then cried out loudly, a sharp rasping cry, then quieted a little; the same thing was repeated in a short time. Her mind was clear. There was no defect of speech.

Head: The head was not enlarged and there was no evidence of injury about the scalp. The eyes were wide open. The pupils were dilated but reacted to light. There was no discharge from the eyes or nose. There was sordes on the teeth.

Neck: A few postauricular glands were palpable on the left side. The neck was rigid. When the head was raised the shoulders and trunk came up also.

Thorax: There was good resonance over the lungs on both sides and the boundaries were normal and mobile. A few moist râles were heard posteriorly.

Heart: Area and sounds normal; no murmurs or adventitious sounds.

Pulse: Soft, easily compressible, and accelerated.

Abdomen: Liver and spleen not palpable, no abdominal tenderness.

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Genitalia: Negative. No enlargement of glands except as above noted.

Extremities: The arms were held in semiflexed position all the time and were kept in chronic clonic motion. The fingers could be flexed and the arms extended, adducted, and abducted, but the child complained of considerable pain whenever they were moved. The back was held rigid. The lower extremities were held in semiflexion. A decided Kernig was present on both sides.

Reflexes: Eyes reacted to light and accommodation. Knee- and Achilles-jerks somewhat exaggerated. Reflexes of upper extremity present.

Blood examination: Reds, 4,900,000; whites, 11,000; hemoglobin, 95 per cent.; index, 0.9.

Differential: Small mononuclears, 13; large mononuclears, 7; polynuclears, 80.

Spinal fluid: Examined Nov. 17, 1910. Amount, 16 c.c., under pressure; looked like water; centrifuged nine hours; a few lymphocytes but no bacteria found.

Urine, negative.

Wassermann reaction, negative.

The temperature on entering the hospital was 100.4 F. rectal; pulse 120.

Following spinal puncture the pulse-rate dropped to 90, to 80, and the rectal temperature remained below 100 F. All symptoms subsided, and in a few days the patient was convalescent.

Diagnosis.—The severe headaches, the rigid neck and back and the presence of Kernig's sign were sufficient to suggest meningeal involvement. She had a general tremor involving the head, trunk and extremities. The tremor was constant. It was greatly increased in voluntary movement or in touching the patient, as she was hyperesthetic. The tremor was coarse, the excursions being large. During movement of the patient the term semi-clonic would better describe it than tremor.

In regard to the lumbar fluid, the stained specimens made after centrifuging the fluid nine hours failed to reveal any organisms, and only a few lymphocytes. This might have been interpreted as a negative finding, or one suggesting a tuberculous or luetic infection. A Wassermann reaction of the patient's blood was negative; consequently lues was strongly improbable.

The patient recovered rapidly following the lumbar puncture, so that tuberculosis could not be considered. There was no infection of the nose, throat, ears or sinuses that could be made out and to which the meningeal symptoms might have been considered secondary. She had what looked like a small burn over the left elbow, but there was no evidence of any considerable infection of it. There was no relation to be made out between this and the meningeal involvement. There had been no injury to the head nor was any intoxicant or narcotic to be considered in the matter.

After being in the hospital several days an examination of a vaginal smear showed it positive for gonococci. She was, therefore, transferred to the children's annex.

While we might speculate on the character of the meningeal involvement, we certainly had no definite point on which to make an etiologic diagnosis, directly or indirectly. It might be consistent for us to consider

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the possibility of serous meningitis, because she presented some of the symptoms of meningitis, because the lumbar fluid was under pressure, because it was practically negative on examination, and because she recovered promptly following puncture. It possibly had some toxic or infectious origin, a view which might be reinforced by the existence of a slight temperature, but we had no leading points on which to base it nor with which to associate it.

CASE 3.—M. L., girl aged 13, Jewish, born in Russia.

Family History.—This was the oldest and only living child. The mother's second pregnancy terminated in a miscarriage at the third month. The third child born was healthy at birth but died at six months of summer complaint. The result of the fourth pregnancy was a dead-born child. No insanity on the father's or mother's side so far as known. Parents were both in good health.

Personal History.—Her early development in regard to walking and talking proceeded about the same as most children. She was bright and as quiet as the average child. The patient had brain fever when 2½ years old and was sick for three months. At six years she had scarlet fever, but recovered entirely. She never seemed to care much for the company of children, preferring to be with older people. She started to school at 8 years but did not learn well.

Last summer (1910) the patient was attacked by four children, who threw her down and beat her. Her cries brought a neighbor, who found her greatly frightened, crying and trembling all over. When the mother returned home in the evening she was still greatly agitated. That night she slept but was very restless. On the following day, Tuesday, she went with other children for a boat ride. On returning home the mother thought she appeared sad. She answered questions and talked voluntarily. Wednesday evening, when getting out of the bath tub, she fell and struck her head. The mother noticed on helping her up that she was very pale, but she complained but little of her head hurting. When the mother returned from her work Thursday she found the patient crying. She said she did not want to live, because the children annoyed her. That night she slept but little, cried a great deal and talked in this same strain.

On the following day she stopped talking and maintained silence for ten days. She would not leave the house, would seldom answer a question, and if so, with a grunt, and would not eat unless her mother insisted on it. At the end of ten days she suddenly became very talkative and noisy; she talked in a rambling way and would repeat almost everything that had been said to her during her quiet spell. She wanted to go to parks, nickel shows, etc., and wanted to become an actress.

Then periods of complete silence and agitation alternated regularly every ten days, for six months. She was taken to a sanatorium, from which she escaped after four weeks. She did not appear to improve while there, but after returning home there was a period of three months in which she seemed to do much better, did not have her mute spells and was quieter in general. While in the sanatorium it was noticed that the spells of silence and agitation were not so uniform. At no time had she had delusions or hallucinations.

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Following the three months improvement, the mother noticed one day while the child was at the piano that a change came over her. She stopped playing and looked strange. On the mother inquiring the cause of the change she said her head hurt. After awakening on the following morning she did not speak for thirteen days. During this time she started to menstruate for the first time. Following a similar alternating period of agitation, she became quiet and during this time she also menstruated. She was subsequently removed to the Elgin Asylum.

Examination.—Lungs, heart and abdomen negative; urine negative.

Nervous System: Head did not show any evidence of past injury. Eyes reacted to light and accommodation.

Muscular System: No paresis or paralysis.

Sensation: No disturbance detectable.

Reflexes: Present but not exaggerated. No hysterical stigmata could be made out.

The family history was strongly suggestive of lues. The girl, however, did not present any evidence of luetic lesions past or present.

The mental change followed closely on the fright and head injury. The alternating periods of silence and agitation followed each other with great regularity. She showed a remarkable memory during her periods of agitation, not only of what was said during her spells of silence, but of early events in her life, of her native city, Odessa, etc. She was also physically restless at these times, while in her periods of silence she would stand in one position for hours.

In view of the clinical history of this case the question of diagnosis would probably consider circular insanity and dementia præcox. The alternating character of the periods of depression and emotional excitement would strongly suggest a circular form of insanity. However, such a picture might occur in the early stage of dementia præcox.

During her periods of depression, there seemed to be a complete suspension of mental activity, lack of interest in her surroundings and no tendency to physical effort, all of which points are strongly suggestive of dementia præcox. At no time, however, had she had the hallucinations or delusions that are frequently present in the beginning of dementia præcox.

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ABSTRACT OF DISCUSSION

Dr. Cassius C. Rogers, Chicago: I have operated on a number of brain tumors and cysts in children with comparatively favorable results, and what I would emphasize in this paper is that children with intracranial surgical lesions should receive the benefit of surgery early before permanent damage has been done to certain organs. These patients with brain tumors or intracranial cysts always have intracranial pressure with the typical symptoms of pressure that cannot be mistaken for anything else; e. g., persistent headache, usually local-

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ized and constant; vomiting without nausea and irrespective of taking food. It is a projectile vomiting. They have impairment of vision on account of the intracranial pressure, and if the pressure is not relieved they have blindness that will ultimately become permanent even after the intracranial pressure is relieved. If the tumor is in the motor area, there is paralysis of one extremity or complete hemiplegia. I have had two patients with brain cyst. One of these patients was 8 years of age and had been treated by a leading pediatrician for four months for gastritis, and then this child, with constant headache, vomiting, almost blind, and who had been unconscious for four days, was brought to the hospital. This patient died, as these patients so frequently do (although I don't know why) of pneumonia. The second was a subarachnoid cyst. The membrane was involved until there was no free circulation over the brain and there was a typical leptomeningitis. These cysts can be diagnosed by the fluoroscope. The fluid will coagulate and is not the cerebrospinal fluid. This was first described by Krause of Berlin. We have many of these cases. We have in our institutions for epileptics and imbeciles, children in whom there is constant irritation in the cranial cavity due to adhesions between the dura and the arachnoid, and we have the cases from intracranial pressure. These children can be relieved of the headache and the vomiting and they can be saved from going blind. Even if one cannot cure them one can benefit them by Cushing's decompression operation. I cannot impress too strongly on the members of this Section who see these children in this pitiable condition, the importance of an early diagnosis and operation; and my theory is (although it may not sound well to the parents) that a child blind, imbecile and suffering is better dead than alive. If there is any hope for them, they should be given the benefit of it, and if they die under the operation it is a justifiable sacrifice.

Dr. William J. Butler, Chicago: Brain tumor, if my experience has been typical, is not particularly frequent among children, or in adults, for that matter, but I believe we could recognize the cases we do see considerably earlier if we would exhaust our resources of information in determining their presence or absence, and among the most important of these resources is the ocular examination. I believe we ought to examine early in a case in which we have reason to suspect a brain tumor, or increased cerebral pressure, for the eye grounds will often show very early changes. That is particularly true in young children. I think it has been an unfortunate thing for those who have been the subject of brain tumor, that they have not been given opportunity for relief; that is, symptomatic relief. I do not know how often we can localize the tumor, and even when found it may be in a location where it would be impossible to remove it; still it is criminal negligence to allow patients to go on suffering when they might be relieved by a decompression operation. In many cases they can be relieved of the certainty of blindness and of the severe headaches and the dizziness that is a marked feature of the condition. In regard to the case with meningeal symptoms, we could not determine the causative factor. I have since had a similar case in which a spinal puncture was followed in twenty-four hours by convalescence.

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RELATIONSHIP OF GASTRIC TO PANCREATIC FAT DIGESTION IN INFANTS

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MINNEAPOLIS

The presence of a ferment in the adult stomach, which has the power of splitting neutral fat into its component fatty acids and glycerin, the gastric lipase, has been confirmed by recent studies.

Although Marcet,[1] Cash,[2] Ogata[3] and Vaughan Harley[4] found fatty acids in the stomach contents, it remained for Volhard[5] to demonstrate the presence of fat-splitting ferment and bring the subject forward for study. Stade,[6] one of Volhard's pupils, confirmed his results and suggested the technic of examination which goes by his name.

Inouye[7] failed to find evidence of a gastric lipase, but Falloise[8] confirmed its presence, and Laquer found the secretion from a Pawlow small stomach to be active in splitting fat, thus proving that the activity cannot be due to regurgitated pancreatic steapsin. Stef. v. Pesthy[9] confirmed the fact that fat is split in the stomach.

The study of the gastric lipase in the infant was first taken up in 1905 and its presence demonstrated by one of us (Sedgwick)[10] and the

*From the Laboratory of Physiological Chemistry, University of Minnesota.

1. Marcet: Med. Times and Gaz., New Series, 1858, xvii.
2. Cash: Ueber den Anteil des Magens und des Pankreas an der Verdauung der Fette, Arch. f. Anat. u. Physiol., 1880, 323.—Cited from v. Noorden's Handbuch der Pathologie des Stoffwechsels, i, 37.
3. Ogata: Die Zerlegung neutraler Fette in lebendigen Magen, Arch. f. Anat. u. Physiol., 1881, vi, 515; cited from Jahrb. d. Fortschr. d. Thierchemie, 1882, xi, 290.
4. Harley, Vaughan: Brit. Med. Jour., 1897, i, 1218.
5. Volhard: München. med. Wchnschr., 1900, 5 and 6.
6. Stade: Untersuchungen über das fettspaltende Ferment des Magens, 1902. Braunschweig, Vieweg and Son.
7. Inouye: Archiv. f. Verdauungskrankheiten, ix, 326.
8. Falloise, A.: Arch. internat. de physiol. iii, 396-497.
9. Pesthy, Stefan v.: Arch. f. Verdauungskr., xii, 292; cited from Jahresb. u. d. Fortschr. d. Tierchemie (Maly), xxxvi, 363.
10. Sedgwick: Jahrb. f. Kinderh., lxiv, 194; Arch. Pediat., 1906, xxiii, 414

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results published in 1906. This work showed that there is a fat-splitting ferment present in the infant's stomach, and in the glycerin extract of the gastric mucous membrane. It was demonstrated that lipase is present during the first few hours of life in the rabbit, and at least as early as on the fourteenth day in the infant. The fat was found to be split to the extent of from 2.9 to 10.6 per cent. in the recovered gastric contents; and *in vitro*, using egg yolk emulsion according to Stade, it was found that as high as 24 per cent. of the fat was split in twenty-four hours. The acids recovered were mostly higher members of the series, insoluble in water and not volatile.

This work on the infants was very shortly confirmed by Heinsheimer.[11] Rietschel[12] did not find any fat-splitting power in the gastric juice from the Pawlow stomach of 8-day-old suckling pigs. Rietschel, however, expressly states that, because of the difficulty in collecting a sufficient working quantity from such small animals, the juice with which he worked was kept some hours or even days under toluol in the ice chest.

Ibrahim[13] found lipase to be absent in fetuses of 150 gm., 250 gm. and 780 gm., but present in small quantities in a fetus of 800 gm., and plainly present in those from 1,100 gm. upwards. The fat-splitting power was very strong in the glycerin extract of the gastric mucosa of a 9-months-old infant which died of pneumonia. Ibrahim's statement regarding the toluol is as follows:

"How can our positive results be brought into line with the negative findings of Rietschel and Engel? We believe it is the toluol that is to blame for the discrepancy. I know perfectly well that Sedgwick and Heinsheimer worked with toluol and got positive results. I suspect that they used but small quantities of toluol. We have been able to show, in a special series of experiments, that the toluol has a markedly inhibitory or completely suspending effect on the activity of lipase from the infant's stomach. It appears to act by an effect on the egg emulsion, for the juice which is freed from the excess of toluol by filter paper possesses fermentive activity. We used thymol. The addition of 2 per cent. sodium fluorid seems also not to be disturbing. We found later also that Laquer has noted this effect of toluol on the gastric lipase. I believe therefore that negative results which are gained in the presence of toluol should be considered of little importance."

11. Heinsheimer: Deutsch. med. Wchnschr., xxxii, 1194.
12. Rietschel: Monatschr. f. Kinderh., vi, 333.
13. Ibrahim: Verhandl. d. deutsch. Gesellsch. f. Kinderh., Koln, 1908, p. 36.

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This has been quoted in full to correct a mistake, and to call attention to an observation published by Sedgwick in 1906, which has not come to Heinsheimer's attention. The statement in Sedgwick's article,[10] that all his experiments were conducted under toluol, is an error which crept in after the manuscript left his hands, as such was not the case. In fact in the *Archives of Pediatrics*[10] the full data of an experiment showing the inhibitory action of toluol are given. Hecht[14] was able also to confirm the presence of a gastric lipase as well as the high per cent. of higher fatty acids which result from its action. The presence of lipase in the gastric juice of infants may therefore be considered to be established.

The presence of steapsin in the pancreatic juice of adults is so well known as to require no further discussion. Pancreatic steapsin has also been found in the infant.

As the gastric lipase acts on emulsified fat (Volhard), and as the food of the infant contains fat in an emulsified state only, the study of the fat ferment becomes of especial importance for the first year of life. It has been stated above that the fat-splitting in the stomach is limited in quantity. Considering these facts we began the study of the relationship between the gastric and pancreatic fat digestion.

In order to obtain gastric lipase entirely free from pancreatic steapsin we used the secretin from a Pawlow small stomach of a dog.[15] This was active, as the following results show:

Egg emulsion, 10 c.c., + thymol in chloroform were left in the incubator at 37 C. for twenty-four hours; 100 c.c. of ether were then added and the whole thoroughly shaken. After standing, 50 c.c. of the supernatant fluid were poured off and after the addition of phenolphthalein, required 1.2 c.c. n/10 NaOH for neutralization. A parallel experiment with the addition of 3 c.c. of the gastric juice from the Pawlow small stomach required 4.5 c.c. n/10 NaOH.

This shows a definite splitting.

In connection with the Schütz-Borissow law that the product of digestion is as the square root of the quantity of ferment, the following is of interest, as indicating that the splitting is due to a ferment. Treated as above:

One c.c. of Pawlow gastric juice split off 2.4 n/10, 4 c.c. of Pawlow gastric juice split off 4.9 n/10, almost exactly twice the amount, or $\sqrt{4}$.

14. Hecht: Wien. klin. Wchnschr., 1909, 1404.
15. We are indebted to Dr. A. E. Wilcox for the skilful operation on the dog.

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Nine c.c. however required but 3 c.c., which may mean that the acidity became so high as to inhibit the action.

The technic of Donath[16] was then used, modified as follows:

Twenty c.c. of neutralized olive oil emulsion were held at 37 C. under varying conditions, as indicated in Table 1, and titrated at the end of stated intervals with n/10 NaOH.

As the experiments recorded in Table 1 show, the attempt was made to subject the emulsion in flask *b* to conditions similar to those in the body—that is, two hours under the influence of gastric juice, then neutralized and digested with pancreatic extract for three hours. The fatty acids produced required 66.1 c.c. n/10 as compared with 53.7 c.c. n/10 required in experiment *c*, which was not acted on preliminarily by the gastric juice. The amount of fat split was 23 per cent. greater when the emulsion was subjected to the gastric lipase before the pancreatic ferment. Control experiment *a*, in which neither ferment was used, could be neutralized by 0.9 c.c. n/10 NaOH. This shows the extent of the splitting due to the ferments. Control *d*, in which the gastric juice alone was used, shows that the inferior and slow action of the gastric lipase alone cannot account for the extent of the splitting.

In Table 2 it is shown that when the digestion is carried on with boiled gastric juice for two hours before the pancreatic extract is added the combined splitting action is lessened.

The two experiments tabulated in Table 3 were carried out as follows:

Egg yolk emulsion was used containing three yolks to 100 c.c. in both. To 10 c.c. of this fat emulsion, flask *a*, 5 c.c. of juice from the small stomach of the dog were added and kept in the incubator with thymol and chloroform for two hours. At the end of this time the digestion was neutralized and 5 c.c. of pancreatic extract were added. Flask *b* was treated in the same manner, with the gastric juice, but the pancreatic extract was omitted. Both flasks were again placed in the incubator for three hours, at the end of which time 100 c.c. of ether were added to each, and both were shaken and allowed to settle. Fifty c.c. of the supernatant fatty acid solution obtained were added to 75 c.c. of alcohol and titrated. Contents *a*, which had been subjected consecutively to both ferments, required 7.3 c.c. n/10 NaOH. Contents *b*, which had been acted on by the gastric ferment alone, required but 3 c.c., less than half as much.

16. Donath, H.: Aktivierung u. Reaktivierung von Pankreassteapsin, Beitr. z. chem. Physiol. u. Path., 1907, x, 390.

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TABLE 1.—EXPERIMENTS TO SHOW THE RELATIONSHIP BETWEEN GASTRIC AND PANCREATIC FAT DIGESTION

20 c.c. of emulsion+

	a	*b*	*c*	*d*
Water	7 c.c.	0	2 c.c.	5 c.c.
Gastric juice from stomach of dog	0	2 c.c. 2 hrs. Neutralized	0 2 hrs.	2 c.c.
Pancreatic extract*	0 5 hrs.	5 c.c. 3 hrs.	5 c.c. 3 hrs.	0 2 hrs.
n/10 NaOH	9 c.c.	66.1 c.c.	53.7 c.c.	1.5 c.c.

20 c.c. of oil emulsion+

*The pancreatic extract was composed of commercial pancreatin and glycerin.

TABLE 2.—EFFECT OF BOILED GASTRIC JUICE ON COMBINED FAT-SPLITTING ACTION

Egg yolk emulsion, 10 c.c.

	a	*b*
Water	3 c.c.	3 c.c.
Juice from small stomach*	2 c.c. 2 hrs. Contents neutralized at end of 2 hrs.	2 c.c. 2 hrs. Neutralized after 2 hrs.
Pancreatic extract	5 c.c. 3 hrs.	5 c.c. 3 hrs.
Titration n/10 NaOH	17.7 c.c.	20.1 c.c.

*Gastric Juice Boiled in *a* but not in *b*.

TABLE 3.—EXPERIMENT IN FAT-SPLITTING USING EGG YOLK EMULSION

Egg yolk emulsion 10 c.c.

	a	*b*
Juice from small stomach	5 c.c. 2 hrs.	5 c.c. 2 hrs.
Water	0 Neutralized at end of 2 hrs.	5 c.c. Neutralized
Pancreatic extract	5 c.c. 3 hrs.	0 3 hrs
First titration: 100 c.c. of ether added to each, *a* and *b*, shaken and settled; supernatant fatty acid solution, 50 c.c. + 75 c.c. of alcohol requires for neutralization, n/10 NaOH.	7.3 c.c.	3.0 c.c.
Second titration of fatty acids from fat not split by the ferment	16.3 c.c.	20.0 c.c.
	23.6 c.c.	23.0 c.c.

TABLE 4.—EXPERIMENTS WITH EGG YOLK EMULSION, USING GASTRIC CONTENTS FROM AN INFANT

10 c.c. egg yolk emulsion+

	a	*b*	*c*
Gastric contents from an infant	5 c.c. In the incubator 2 hrs.; alkalinized*	5 c.c. As *a*	0
Pancreatic extract	0 3 hrs. longer at 37 C.	5 c.c. As *a*	5 c.c.
100 c.c., ether added and titrated and as in Table 3—			
First titration n/10 NaOH	2.2 c.c.	6.8 c.c.	3.8 c.c.
Second titration n/10 NaOH (after saponification)	35.0 c.c.	30.2 c.c.	
*Neutralized + 2 c.c. NaOH.	37.2 c.c.	37.0 c.c.	

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The experiments in Table 4 were carried on as in Table 3, using gastric contents from an infant instead of the gastric juice from the small stomach of the dog. The egg emulsion digested with 5 c.c. of gastric contents for two hours, neutralized, and then rendered alkaline by the addition of 2 c.c. n/10 NaOH, and digested further for three hours, after being treated with ether and alcohol as before, gave a titration of 2.2 c.c. n/10.

When the pancreatic extract was added after the two-hour digestion with gastric ferment the titration was 6.8 c.c. n/10. When digested three hours with the pancreatic juice alone the titration was 3.8 n/10.

Gastric digestion alone five hours	2.2
Pancreatic digestion alone three hours	3.8
	6.0
Gastric digestion two hours alkalinized + pancreatic digestion three hours	6.8

From the above we feel warranted in concluding that the neutralization of the fat gastric digestion does not end the function of the gastric lipase of the dog or infant. Whether the effect shown is a summation of, or a relationship between, the actions of the two ferments, preparatory or activating, our results do not permit us to state with certainty. It seems justifiable, however, to conclude that the function of the gastric lipase of the infant is not ended when the gastric contents pass the pylorus and are neutralized in the duodenum.

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THE RELATION OF MEAT INGESTION TO INDICANURIA IN CHILDREN

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SAN FRANCISCO

With the view of gathering, if possible, some data on the use and abuse of meat diet in childhood, the work reported in this paper was begun about a year ago. It was hoped that the results of the experiments might lead to some definite indications as to the quantity and frequency with which this article of diet should be allowed.

So much work has been done on the dietetic requirements of infancy, and from both theoretical and practical standpoints so many valuable conclusions have been drawn, that it seems strange that more work has not been done to settle the same problems in older children.

The insidious, often marked, symptoms that chronic dietetic disturbances show after two years of age, compared with the more acute signs of intestinal indigestion in infancy, probably account for the rather neglectful attitude that even pediatrists have taken toward regulating the diets of older children.

If one makes it a practice to discuss with his patients their attitude towards the giving of meat to children, he will find as a rule that one of two rather disagreeable conditions exists. Either the child receives no meat at all, the parents laboring under the only too frequently met delusion that meat soups contain all of the valuable constituents of the food, or that the child receives meat twice or even three times a day. It is a question which of these two evils is the worse. The one that puts on the kidneys the duty of excreting a wealth of useless extractives, or the one which forces on the youthful organism three meals a day—more than sufficient to supply the caloric needs of a working adult. One thing is definite, however, and that is that if the next generation of men and women is to be a healthy one we must so regulate the conditions under which the children live that their growth may not be impeded by chronic intestinal disturbances.

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During the past two years a large number of stools has been examined incidental to routine investigation, and while it has been strikingly common to find free starch and free fat, especially in those children with clinical symptoms of intestinal disturbance, it has been rather surprisingly rare to find undigested muscle-fiber, except in the most marked cases. Bearing this in mind, and desiring, if possible, to reach some conclusion as to the advisability of giving meat to children over two years of age, it was decided to investigate the pathologic products which the urine might contain, which might have some bearing on this subject.

Taking into consideration that an excess of meat in the diet would probably lead to improper digestion and consequent putrefaction, it seemed reasonable to assume that the amount of indican in the urine of children fed on small and large quantities of meat might throw some light on the subject.

Appreciating the fact that under normal circumstances little or no indican is excreted in the urine, it seemed possible that if meat were given in excess, digestion would be sufficiently interfered with to allow the putrefactive bacteria to act on the excess of protein, and the indicanuria would be a gauge as to the ability of the organism to properly handle the animal food.

It is almost universally conceded now, as Nencki and others have shown, that indol, the forerunner of indican, is produced in the intestine solely by bacterial putrefactive decomposition of the protein food. There have been a few dissenters from this view, notably, Flint, who states that it may be formed by the action of the tryptic ferment of the pancreatic juice on the protein food. Herter states that such a phenomenon could only occur in the intestines in the presence of excessive amounts of protein food combined with greatly delayed digestion. Ordinary absorption occurs in the peptone and amino-acid stage of proteolytic digestion and it is hardly conceivable that a rapidly proteolyzed meal should lead to the formation of indol. In unmistakable and most conclusive support of the bacterial putrefactive source of indol, stands the work of Senator, who in a large number of investigations failed to detect the merest trace of indican in the urine of new-born babies, the stools of which are always bacteria-free.

The object in selecting indican as the urinary by-product, which might throw some light on the use of meat diet in childhood, was two-

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fold. First, because it has been definitely proved that one of the chief causes of indicanuria is an excess of food, which by hampering digestion allows the putrefactive bacteria to work, and second, because it seemed advisable, if possible, to have some single test that might be of use to the ordinary practitioner.

That accurate nitrogen and sulphur determinations may in the future throw some light on this very interesting subject is probable, but this does not come within the province of anyone but the trained chemist and is therefore of little value in aiding the clinician.

The minute details of a well-planned piece of investigation which one can carry out successfully in a properly organized laboratory, are not within the power of the best intentioned worker when he has to deal with institutional children as the objects of his experiments; and when, furthermore, he has to depend on the gracious willingness of institutional attendants to carry out instructions as to diets, he is truly in a dilemma that is discouraging. On account of this difficulty, which is practically unsurmountable, it seemed advisable to pursue the work without disturbing the routine of the home from which the specimens were obtained, knowing that quantitative diets could not be given, and that only untrained assistants would have to be depended on to furnish the daily specimens of urine. The diets given to the children corresponded to the diets usually given to children in the ordinary orphanages and no attempt was made to modify this under the first series of tests that was carried out.

A brief résumé of this diet may be *apropos* in order that one may understand fully the most important factor in determining the conclusions. For breakfast: fruit, cereal, toast and milk. Dinner: milk soup, meat, potatoes, green vegetable, and dessert. Supper: bread, milk and fruit. The diets were somewhat variable as to the particular kinds of cereals, meats and vegetables, but otherwise were fairly constant.

With the object in mind of determining the influence which age had on the ability to digest meat, it was decided to divide the children into four groups. In the first group were six children 12 years of age. In the second group, six children 9 years of age. In the third group, six children 6 years of age, and in the fourth group, six children 3 years of age. The early morning urine was the sample tested in each case and the Obermayer test was the one employed in formulating the results.

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A rather arbitrary method was used for designating the quantity of indican present in each specimen, + representing a moderate reaction, + + a marked reaction, and + + + a very marked reaction. The children on whom the tests were made were normal children as far as physical examination could determine. In each case the urine was carefully examined in order to exclude pathologic conditions of the kidney, and no child was observed in whom the urine showed the slightest deviation from the normal.

In the first group of children tested were six normal girls, 12 years of age, in each of whom twenty-five specimens of urine were examined, making a total of 150 experiments, in fifteen of which on different days a single + reaction was obtained. In other words, 10 per cent. of the specimens of urine of 12-year-old institutional children showed a moderate reaction for indican, indicating a slight amount of intestinal putrefaction with the diet which they were receiving.

In the second group of children were six girls 9 years of age. One hundred and fifty specimens of urine were tested and twenty of them showed a single + reaction with the routine diet, a slight increase (13 per cent. as compared to 10 per cent.) over the number of positive reactions among the 12-year-old children but not sufficient to warrant any deduction.

In the third group of children were six girls 6 years of age. One hundred and fifty specimens of urine were examined and in about 15 per cent. of the examinations a single + reaction was obtained.

In the fourth group were six children 3 years of age, and the results here were decidedly interesting because they showed that in children of this age the amount of protein food received in an ordinary diet containing milk and meat once daily gave rise to considerable intestinal putrefaction and indicanuria. Twenty per cent. of these examinations showed a moderate single + reaction and 15 per cent. gave a marked + + reaction. In all of the cases under observation, one or more normal bowel movements occurred every day, so that constipation could in no way have had an influence on the results.

These experiments, which were made with the children receiving ordinary diets, were now repeated after the children had been given meat twice daily for two weeks. The instructions given were that the children under observation should receive the same diets as previously,

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except that they should receive in the evening the same quantity of meat as at noon. In the 150 tests made on the urine of the 12-year-old girls, 2 per cent. showed a marked double + + reaction and 12 per cent. showed a moderate single + reaction. The results were practically identical with those obtained when the children were getting meat but once daily. In the 150 tests made in the second group of children 9 years of age, 14 per cent. showed a moderate single + reaction, these results also being practically the same as when the children were getting meat only once daily.

In the observations made on the 6-year-old children who were receiving meat twice daily, the results were rather more striking. Twenty-five per cent. showed a moderate single + reaction for indican and 15 per cent. showed a marked double + + reaction. This was a decided increase in the number of positive reactions—25 per cent. of moderate single + reactions as compared with 15 per cent. and 10 per cent. of marked double + + reactions as compared with no + + reactions when the children were receiving meat but once daily.

In the 3-year-old children the results were also very striking. Thirty-five per cent. of the reactions were single + or moderate reactions and 30 per cent. were double + + or marked reactions as compared with 20 per cent. of moderate reactions and 15 per cent. of marked reactions in these same children when they were receiving meat but once a day.

The results, while strongly suggestive, and while warranting one in advocating certain principles in the feeding of children, might be criticized on the ground that the conclusions are not based on the observation of a sufficiently large number of children. Admitting this, still the absolutely negative results in the 9- and 12-year-old children, with the positive results in the 3- and 6-year-old children are very suggestive.

A brief résumé of the results obtained shows as follows:

1. With an ordinary diet containing meat once daily, children 6, 9 and 12 years old succeed in digesting their food fairly well without much putrefaction in the intestinal tract. Three-year-old children, on the other hand, with the same diet are much more apt to show indicanuria.

2. The giving of meat twice daily produces in 9- and 12-year-old children practically no change in the amount of intestinal putrefaction.

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3. In 6- and even more markedly in 3-year-old children meat given twice daily gives rise to a decided increase in the indicanuria and is a cause of pronounced intestinal putrefaction.

4. As limited as these observations may be, they seem to point to one definite conclusion, and that is that the giving of meat more than once daily to children under 9 years of age is a practice conducive to intestinal putrefaction and indicanuria, and is to be deprecated on account of the harm resulting to the organism from the constant presence of abnormal products of putrefaction.

350 Post Street.

ABSTRACT OF DISCUSSION

Dr. J. P. Sedgwick, Minneapolis: I was interested to note the evident care which Dr. Fleischner has taken in going through this work. This is so much more important in children, as indicanuria is not so common as in adults. The use of some quantitative method of estimation, such as Folin's, would have added value to the work by making it possible for others to compare their results with Dr. Fleischner's.

Dr. L. Porter, San Francisco: We have found the work of Dr. Fleischner of great value to us. It demonstrates that when a little child is very irritable its urine will show indican and that we can prognosticate from a heavy indican reaction an irritable, easily fatigued child.

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EPIDEMIOLOGIC AND ETIOLOGIC STUDIES OF ACUTE POLIOMYELITIS IN PENNSYLVANIA

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During 1907 some 200 cases of poliomyelitis developed in a district in the northwestern section of Pennsylvania, extending from 78° 55′ to 80° 15′ west longitude and from 41° to 41° 30′ north latitude. The outbreak apparently began the latter part of July in Elk County and in early August in Venango County, appearing first in Elk County at Ridgway and in Venango County at Oil City; later at Du Bois, in Clearfield County and at Eau Claire in Butler County. In this outbreak the height of the epidemic seemed to be reached the latter part of September and by the end of November the disease was practically stamped out. A small outbreak occurred in Adams County during July and August, 1908. Sporadic cases, only, occurred during 1909.

During the summer of 1910 an epidemic of considerable proportions developed in widely separated sections of the state, reaching a total of 1,076 cases before dying out in the early winter months. The 1910 epidemic began in June in the Lehigh Valley, in July in Lancaster County and in Potter County, and later small outbreaks developed in Westmoreland and Fayette Counties, with scattering cases throughout fifty-five counties. In the Lehigh Valley in the vicinity of the two Bethlehems, Allentown and Catasauqua, the outbreak assumed rather alarming proportions and the same may be said of Lancaster County. In Philadelphia the greatest number of cases occurred in a beautiful section of Germantown, in the northern part of the city, and in a new and rather open section of West Philadelphia, with well paved streets and plenty of shade.

The 1,076 cases reported during the year 1910 were grouped geographically with reference to topography, showing conclusively that

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elevation played no part, as cases occurred at Philadelphia near sea level in considerable numbers and others were reported in areas fully 2,500 feet above the level of the sea. Similar charting was made with reference to distribution of surface waters, with negative results. The only features of this part of the analysis worthy of mention are that moisture and possibly open water-courses may have played some part in the distribution of the disease.

In classifying cases by geologic formation it seems to hold true that over sand and gravel formation the disease was less prevalent and that it was more prevalent in districts not so favorable for drainage. The character of the surface of the earth in each locality and of the subsoil seems to have been worthy of special note in this relation; and while it is of no distinct value in relation to our present knowledge of the disease, it may be worth recording when making future studies.

We were able to learn nothing of value in relation to the distribution of vegetation other than that the disease was most prevalent after vegetation had reached its height and during the stage when full development of plant life had been reached or passed, the greater number of cases resulting when plant life was on the decline and about the time the harvest season had ended. Possibly fungus growths favored by local death of plants may have furnished a nidus for unusual development of an intermediate host in which the virus may have passed through some stage of its life cycle.

Seasonal distribution shows that the disease is most prevalent during the height of summer and early autumn. This distribution is well shown by grouping reported cases by months of sickening, as follows:

	Cases
January	0
February	3
March	2
April	1
May	4
June	27
July	162
August	336
September	318
October	152
November	52
December	10
Month of onset indefinite in	9

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During the 1907 epidemic, the chief of the laboratories spent considerable time in the infected area and secured pathologic materials for laboratory study. Full details of this work will be found in the Department's Annual Report for the year 1907. Research work begun at that time has been continued with slight interruption until the present time. It is doubtful whether monkeys inoculated at that time developed a true poliomyelitis; probably the virus secured was not fresh enough for research work. Bacteriologic studies made at that time were not productive of results.

With the beginning of the 1910 outbreak a branch laboratory was opened in Bethlehem through the cooperation of St. Luke's Hospital, and later a second branch laboratory was opened in the city of Lancaster through the courtesy of the city chemist. A great mass of data was secured, and a number of autopsies were performed. The tissues, together with a number of lumbar punctures, gave us constant material for laboratory study, and as soon as good strains of virus were secured inoculation experiments gave us the disease in considerable numbers of cases in artificially infected monkeys.

In addition to making careful studies of tissues and spinal fluids secured from the bodies of those suffering from the disease and in making careful inoculation experiments with these materials, a vast amount of work was done in studying smears of blood and of nasal mucus. Careful studies of bowel contents and urine were carried out, an offer being made to the physicians throughout the state to study all such materials forwarded to the laboratory, together with an offer to send them shipping containers. It is very gratifying to report that the laboratory staff in carrying out this work have confirmed the published researches on poliomyelitis and our hopes at one time were greatly encouraged by a rather constant finding of a microorganism which we were unable, until recently, to grow in pure culture, in the blood of children suffering with the disease and in the blood of monkeys suffering from this disease.

Details of these reports were published from the laboratory in March, 1911. At that time we claimed nothing more than that the organism was found with great regularity in the stained blood of human and experimental poliomyelitis. The only thing new to add concerning it at this time is its demonstrated motility. We are continuing work with a series of experiments and continually propagating several strains of virus with the hope that at some time we will learn what its etiologic significance may be.

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RESULTS OF INTENSIVE STUDY OF 748 CASES

An intensive study was undertaken by the department, resulting in a more or less complete record of 748 cases, giving details of physical conditions of the premises in which the patient sickened, the history of any possible exposure either to human cases of this disease or to domestic animals that may have had paralysis, the notes being so complete as to include possible exposure to flies, ticks, mosquitos, ants or any biting insect; exposure to heat, cold or dampness; exposure through swimming, wading or paddling and exposure to dust. A detailed clinical record was secured from the greater number of these cases giving the duration of the fever, the average range of fever, pulse-rate, date of onset of paralysis; full notes as to other cases in the family or neighborhood, a note of the extent of the paralysis at the height of the disease, and finally a note of the extent of residual paralysis existing several months after all symptoms of acute illness had subsided.

We are very greatly indebted to upward of 600 physicians practicing in the commonwealth for all of these detailed records, the compilation of which would not have been possible but for their loyal cooperation.

Detailed epidemiologic records were forwarded by 605 physicians, covering 748 cases.

528	physicians	reported	1 case	each
45	physicians	reported	2 cases	each
17	physicians	reported	3 cases	each
7	physicians	reported	4 cases	each
4	physicians	reported	5 cases	each
1	physician	reported	6 cases	
2	physicians	reported	8 cases	each
1	physician	reported	10 cases	

A few interesting observations may be summarized from the many points developed in this intensive study.

Out of 773 cases of poliomyelitis, fifty-nine gave a distinct history of exposure to previous cases, the sexes being equally divided. Nationality seems to play no particular part in this disease, the vast majority of those contracting poliomyelitis being American-born. Males have more frequently contracted the disease than females. Out of this series of cases 437 were males, and 336 were females. But three of the colored race (two males and one female) sickened. In forty-four instances secondary cases developed in the household.

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Other acute illnesses two months prior to the development of infantile paralysis seemed to show but little influence. In three instances whooping-cough existed; in three, intestinal indigestion; in five, measles; in ten, diarrhea; in thirteen, gastro-enteritis; in four, influenza; in nine, tonsillitis; in four, acute enteritis.

Great exposure to heat, cold and dampness was noted in comparatively few instances. Great heat was recorded in sixty-five cases; exposure to cold in twenty-seven; exposure to dampness in eighty-nine.

A total of thirty patients are reported to have met with some minor accident a short time previous to the onset of poliomyelitis.

It was somewhat surprising to find over-exertion reported so seldom, a total of twenty-eight cases being recorded, twenty in the male sex, the exertion often consisting of hard work in the open field.

In nineteen cases a history of automobile riding was noted; in sixteen, swimming; in twenty-nine, wading in open streams; in thirty-seven, paddling in water; in 157 instances the individual contracting the disease constantly ran barefoot.

The prevalence of other diseases in the community during the outbreak of poliomyelitis was not as great as has been reported by some authorities. The diseases of particular moment frequently recorded in infected communities were tonsillitis twenty-five times, gastro-intestinal disturbances forty-seven times.

The date of onset by months is of peculiar interest. Occasional deaths from this disease were recorded during the early months in the year, one in January, two in February and two in April. The disease apparently became more frequent the latter half of June, in each instance in communities where patients had died from the infection at some time during the winter months. In June twenty-four cases were reported; in July 120; in August 262; in September 230; in October 130; in November forty-one; in December ten; a total of 1,076 cases being reported to the department during the twelve months.

By action of the Advisory Board of the State Department of Health, poliomyelitis was made reportable Jan. 7, 1910, general advertisement of the regulation taking place immediately, but individual notices were not mailed to physicians until July, so it is possible that some cases were not reported in the first half of the year. A total of 240 certificates were filed with the department giving poliomyelitis as the cause of death. This mortality shows the disease to have been unusually virulent, even grant-

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ing that a good many cases may have been missed because it had been so recently made reportable.

The average duration of the fever was about four days. The series of cases studied carefully from the family physicians' notes show that in fourteen cases the fever lasted less than twenty-four hours; in thirty-five, two days; in sixty-eight, three days; in sixty-five, four days; in seventy-two, five days; in thirty-four, six days; in 137, one week and over; in eighty cases, two weeks and over.

The temperature range averaged 99 to 104 + F., the records in twelve cases showing 99 to 100; in fifty-four it was 100 to 101; in 106 cases, 101 to 102; in 147 cases, 102 to 103; in 119 cases, 103 to 104; in sixty-five cases, 104 and over.

The pulse-rate was increased in a large percentage of the cases, the average rate being near 100. In twenty-two instances a record is made of a rate above 150. In a vast majority of the cases the pulse tension is given as high.

In tabulating the symptoms present during the onset and early stage of the illness we find irritability present in 372 cases; stupor in 225; nausea in 343; vomiting in 366; retraction of head in 267; retraction of back in 192; nervous twitchings in 345; convulsions in sixty-one; diarrhea in 159; constipation in 349; retention of urine in 118; incontinence of urine in ninety-seven; insomnia in 291; sleepiness in 244, and sore throat in 101 cases. These symptoms seem to apply with great uniformity to both sexes.

Skin eruption was recorded in sixty-three instances. A scarlatiniform erythema was described several times.

It is worthy of note that the presence of pain and tenderness was recorded in 564 cases of the 773 studied intensively. In a very large majority of the cases pain was most severe in the area of the body later showing paralysis and the pain usually subsided on the appearance of paralysis. A greater number of patients complained of pain in the spinal region alone than in any other area of the body, a total of sixty-nine having pain in this region only. Fifty-one complained of spinal and lumbar pain, and in addition, of pain in all extremities. Thirty-three complained of pain in the lumbar region only, nineteen of pain in the cervical region and a total of thirty-seven complained of pain in the large joints, shoulders, hips or elbows; two complained of it in the left thigh only, three in the right thigh, one in the left hand, one in both feet and

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one in the right foot only. Abdominal pain was not frequent except as associated with nausea and vomiting.

In a few instances (thirty-four) evidence of insect bites was noted. Particulars of these marks will be found in another part of the paper.

In this series of 773 cases, 142 patients (18.4 per cent.) recovered without paralysis and 294 (71.6 per cent.) recovered with considerable residual paralysis.

The distribution of paralysis when at its height presents some interesting features, the following table showing how frequently the extremities alone were involved:

Extremity	Times involved	Extremity	Times involved
Left arm only	16	Both legs only	120
Right arm only	20	Left hemiplegia	35
Left leg only	74	Right hemiplegia	42
Right leg only	69	All four extremities	78
Both arms only	4		

In addition to the seventy-eight instances in which the four extremities were involved, additional parts were paralyzed as follows:

All extremities and both eyes	Once
All extremities, right eye and right face	Once
All extremities, left eye and neck	Once
All extremities, back and abdomen	Once
All extremities, left face, left eye, neck, back and abdomen	Once
All extremities and both eyes	Once
All extremities, neck, back and abdomen	Once
All extremities, neck, back, abdomen and left face	Once

In addition to the 120 cases having both legs only paralyzed, the following cases had additional parts involved:

Both legs and right face	Once
Both legs, right arm and abdomen	Once
Both legs, back and abdomen	Once
Both legs, right face, right eye and right forearm	Once
Both legs and right arm	3 times
Both legs and left arm	Once
Both legs, right arm and right face	Once
Both legs, right arm and neck	Once
Both legs, right arm, right eye and right face	Once

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Crossed paralysis was found as follows:

Right upper extremity and left lower extremity	6 times
Left upper extremity and right lower extremity	5 times

In thirty-four instances one or both legs were paralyzed, either alone or in combination with some other part of the body.

In nineteen the right face was paralyzed twice with only the eye or eye and neck involved, and seventeen cases in connection with some other parts of the body.

The left face was involved in twenty-two cases, always in connection with some other part of the body.

Impairment of intelligence was noted in twenty-nine instances.

Housing conditions show that in 149 instances the individual sickened in a large home; in 219 instances in a medium sized home, and in 164 instances in a small dwelling.

When classified with reference to the number of members of the household the tabulation shows two instances in a family of one; thirteen in a family of two; eighty-six in a family of three; 154 in a family of four; 121 in a family of five; ninety-five in a family of six; sixty-two in a family of seven; thirty-three in a family of eight; twenty-three in a family of nine; twenty-three in a family of ten and over.

With reference to the number of children in the household, the tabulation shows 106 instances in which there was one child; 174 in which there were two children; 110 in which there were three children; ninety in which there were four; fifty-nine in which there were five; twenty-two in which there were six; seventeen in which there were seven; twenty in which there were eight; five in which there were nine; four in which there were ten and over; eighteen in which there were none, with 148 unstated cases.

Out of 773 cases contact occurred as follows:

Certain direct contact with acute case	59
Certain direct contact with abortive case	7
Certain direct contact with chronic case	2
Certain direct contact with acute case by second person	5
Adults exposed escaping paralysis	1,678
Children exposed, escaping paralysis	1,062
Children sleeping in same bed room with acute case, escaping paralysis	261

These figures establish the fact that the danger of transmission from one member of the household to another member of the same household

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is not great and when we consider the number of instances in which other children slept in the same bed-room with the child contracting poliomyelitis, it seems still more remarkable that so few secondary cases occurred. In 289 instances children slept in the same bed-room, only twenty-four contracting the disease.

In nine instances the disease developed in children after having visited communities where this disease was known to be prevalent. In five cases the possibility of transmission through a second individual seemed probable. These will be given in detail with the case history. In a number of instances suggestive illness occurred in the same household with frank cases of paralysis. A few such cases will be cited with their histories.

Further studies of household conditions showed that in 390 homes doors and windows were screened. In 128 homes sanitary conditions are classified as excellent; in 215 homes as good; in 226 as fair; in sixty-six as bad, with 138 unstated.

Mosquitoes were reported as having access to the house 324 times.

The dwelling stood near a stable or barn in 297 instances; near a poultry house 289 times; near a privy 275 times; and forty-seven times a public garbage dump is reported near the residence. Stagnant water nearby is reported seventy-five times; near open streams in 171 instances; near open drains in 140 instances.

In 410 instances trees are reported in the immediate vicinity of the dwelling; in 122, shrubbery. The particular trees enumerated will be found in a table.

Sanitary plumbing is reported in 277 homes; outside closets in 476.

Household garbage was disposed of to hogs on the premises in thirty-six instances; was burned in thirty-eight; a public system of disposal was reported 231 times; was buried in four instances; thrown on surface of ground 162 times; fed to other domestic animals eleven times.

The water-supply to the household was given as public in 327 instances; well in 194 instances; spring in sixty instances and cistern in thirty-three instances.

The ice-supply was given as natural in eighty-four instances; artificial in 164 instances; none used at all in 322 instances; with 203 unstated.

The milk-supply was given as one cow in 167 instances; breast-fed in twenty-five instances; condensed in five instances; in bottles in 194 instances; in cans in 112 instances; in own receptacles in twenty instances.

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Fruits and vegetables used prior to illness in the various households and partaken of by those ill may be well summarized in the following table:

Common varieties	117	Watermelon	2
Cabbage	15	Canteloupe	3
Potatoes	19	Pineapple	1
Lima beans	4	Grapefruit	1
String beans	6	Bananas	51
Peas	10	Oranges	47
Parsnips	1	Peaches	36
Carrots	1	Apples	115
Tomatoes	28	Green apples	1
Corn	8	Plums	20
Parsley	1	Grapes	14
Cucumbers	3	Green grapes	2
Celery	7	Cherries	15
Radishes	9	Berries	21
Onions	17	No vegetables used	116
Lettuce	18	Not stated	55

Vermin were reported as follows:

	Houses
Flies were present in	383
Mosquitoes were present in	212
Mice (house) were present in	149
Rats were present in	77
Ants were present in	85
Roaches were present in	90
Bedbugs were present in	91
Spiders were present in	127
Mice (field) were present in	43
Biting flies were present in	169
Ticks were present in	14

TABLE 1.—POLIOMYELITIS GROUPED BY AGE PERIODS

	Cases		Cases
1 month	2	3 to 4 years	108
2 months	1	4 to 5 years	58
3 months	4	5 to 6 years	44
4 months	4	6 to 7 years	50
5 months	7	7 to 8 years	24
6 months	9	8 to 9 years	22
7 months	12	9 to 10 years	20
8 months	7	10 to 15 years	45
9 months	20	15 to 20 years	23
10 months	7	20 to 25 years	18
11 months	8	25 to 30 years	8
1 to 2 years	346	30 years and over	8
2 to 3 years	187	Not stated	34
		Total	1076

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Fig. 1.—Photomicrograph of pure culture of microorganisms found in the blood of patients with acute anterior poliomyelitis, showing long and short forms of the organism.

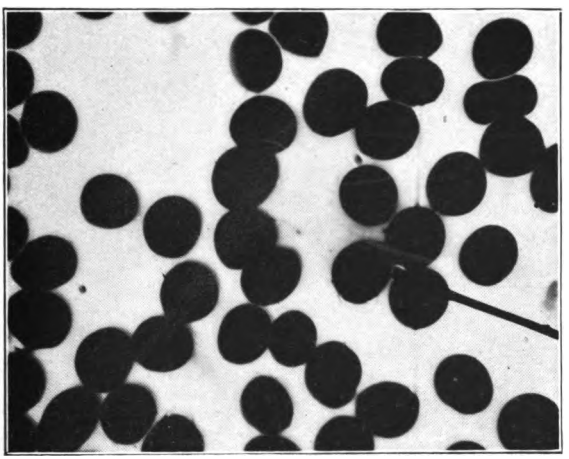

Fig. 2.—Photomicrograph of blood of patient ill of acute anterior poliomyelitis, showing a single organism with bulbous ends connected by a long thin body, attached at one end to red corpuscle in exact center of photograph.

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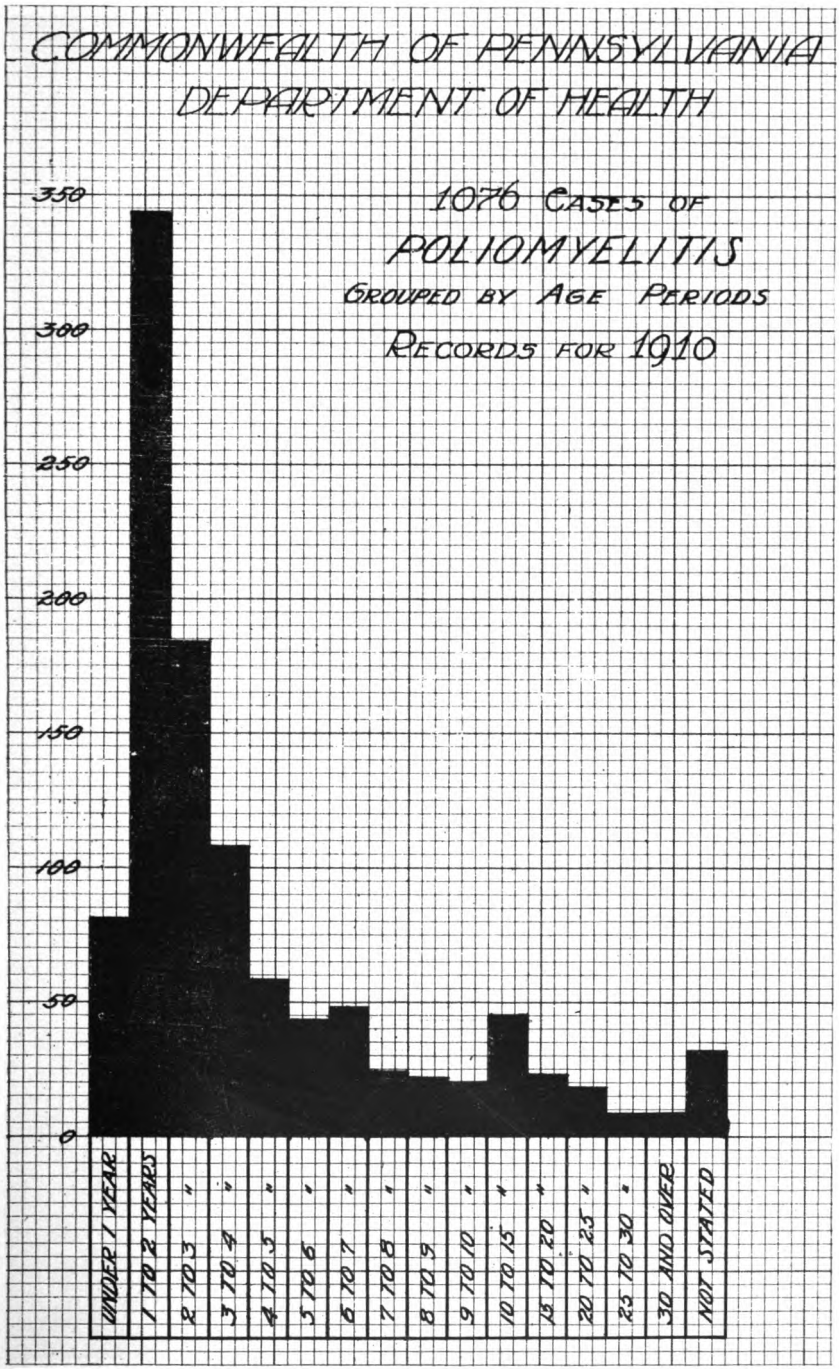

Fig. 3.—Chart showing graphically the cases of poliomyelitis recorded in 1910, grouped by age periods.

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TABLE 2.—DAY OF ONSET OF PARALYSIS

	Cases		Cases
Same day	111	10 days	8
1 day	83	11 days	4
2 days	88	12 days	2
3 days	93	13 days	0
4 days	51	14 days	3
5 days	37	15 days	2
6 days	22	19 days	1
7 days	22	20 days	1
8 days	9	21 days	5
9 days	5	Not stated	126

Trees and shrubbery stood near the infected house as follows:

Trees		Shrubbery	
Fruit	168	Roses	26
Maple	150	Grape Vine	10
Poplar	30	Lilacs	8
Oak	21	Honeysuckle	6
Pine	11	Hedge	3
Willow	9	Vines	1
Locust	9	Snow-balls	1
Chestnut	9	Locust	1
Horse-chestnut	9	Japanese hop vine	1
Elm	4	Cedar	1
Beech	4	Berry	1
Walnut	3	Scrub-apple	1
Linden	2	Maple	1
Cottonwood	2	Mock-orange	1
Cedar	2	Clematis	1
Birch	1	Underbrush	1
Spruce	1	Privet and thorn hedge	1
Shumac	1	Kind not stated	52
Ash	1		
Kind not stated	23		

DATA AS TO DOMESTIC ANIMALS

240 homes had dogs without sickness
264 homes had cats without sickness
254 homes had chickens without sickness
57 homes had birds without sickness
180 homes had horses without sickness
155 homes had cows without sickness
1 home had 1 dog with sickness
3 homes had chickens with sickness (paralysis)
1 home had 1 bird with sickness (paralysis)
1 home had 1 horse with sickness
1 home had 2 cats with sickness

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Insect Bites

Mosquito bites appeared in 15 cases as follows:

Back	1	Body	1
Hands	3	Thighs	1
Face	4	Chest	1
Neck	3	Location not stated	1

Bedbug bites appeared in 2 cases as follows:

Thighs	1	Chest	1

Unknown insect bites appeared in 17 instances as follows:

Face	4	Thighs	2
Neck	2	Temporal region	1
Body	4	Location not stated	3
Head	1		

In many histories rats were reported on the premises. Field mice were frequently found. In 149 homes house mice were prevalent; in 383 homes ordinary flies were found; biting flies were reported 169 times; bedbugs ninety-one times; roaches ninety times; ticks fourteen times; ants eighty-five and spiders 127. Mosquitoes were noted in 212 instances.

Unusual insects were reported in eighteen instances, particulars being found elsewhere.

In three instances paralytic disease occurred in animals, the particulars being set forth elsewhere.

The underclothing worn by those sickening is reported as being cotton in 304 cases; woolen in eighty-two; half cotton and half wool in ten. The stockings were reported as being cotton in 300; woolen in seventy-eight; half cotton and half wool in six.

Several centers of unusual prevalence were noted, one in the vicinity of Galeton near the line between Potter and Tioga Counties, at a great elevation in the northern part of the state; one in the vicinity of the Bethlehems and Allentown in Northampton and Lehigh Counties in the east central section of the state; one in Lancaster County in the city of Lancaster and adjacent rural districts in the south central part of the state and one each in Luzerne and Westmoreland Counties in the southwestern section of the commonwealth, and in Philadelphia county in the section of the city known as Germantown and in a part of West Philadelphia west of the Schuylkill River. The disease was found, however, in fifty-eight counties out of sixty-seven. Grouped by counties the following table shows the distribution throughout the state:

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TABLE 3.—SHOWING DISTRIBUTION OF CASES AND POPULATION

County	Cases	Population	County	Cases	Population
Adams	0	34,319	Lancaster	150	167,029
Allegheny	89	1,018,463	Lawrence	4	70,032
Armstrong	7	67,880	Lebanon	3	59,565
Beaver	17	78,353	Lehigh	101	118,832
Bedford	1	38,879	Luzerne	15	343,186
Berks	26	183,222	Lycoming	4	80,813
Blair	0	108,858	McKean	15	47,868
Bradford	4	54,526	Mercer	2	77,699
Bucks	27	76,530	Mifflin	4	27,785
Butler	14	72,689	Monroe	6	22,941
Cambria	3	166,131	Montgomery	33	159,590
Cameron	2	7,644	Montour	1	14,868
Carbon	10	52,846	Northampton	140	127,667
Centre	4	43,424	Northumberland	7	111,420
Chester	11	109,213	Perry	5	24,136
Clarion	0	36,638	Pike	1	8,033
Clearfield	1	93,768	Potter	9	29,729
Clinton	0	31,545	Schuylkill	15	207,894
Columbia	7	48,467	Snyder	1	16,800
Crawford	1	61,565	Somerset	7	67,717
Cumberland	4	54,479	Sullivan	0	11,293
Dauphin	11	136,153	Susquehanna	0	37,746
Delaware	16	117,906	Tioga	19	42,829
Elk	2	35,871	Union	1	16,249
Erie	8	115,517	Venango	4	56,359
Fayette	19	167,449	Warren	1	39,592
Forest	0	9,435	Washington	21	143,680
Franklin	6	59,775	Wayne	3	29,236
Fulton	0	9,703	Westmoreland	50	231,304
Greene	1	28,882	Wyoming	2	15,509
Huntingdon	1	38,304	York	6	136,405
Indiana	5	66,210	Philadelphia	143	1,549,008
Jefferson	2	63,090			
Juniata	0	15,013		1076	
Lackawanna	4	259,570			

It is interesting to note that few cases developed in Elk, Venango and Clearfield Counties, where epidemics were reported in 1907, and in Adams County, where a small outbreak occurred in 1908. The exception, however, occurred in Butler County, where a few cases developed in 1907, and fourteen cases in 1910.

The laboratory staff, working from the branches opened in the field, made an intensive study of seventy-three cases during life and a study of pathologic materials from eleven fatal cases, doing a great deal of routine work resulting in statistics too elaborate to refer to more than incidentally in this paper. A composite table of blood-counts alone,

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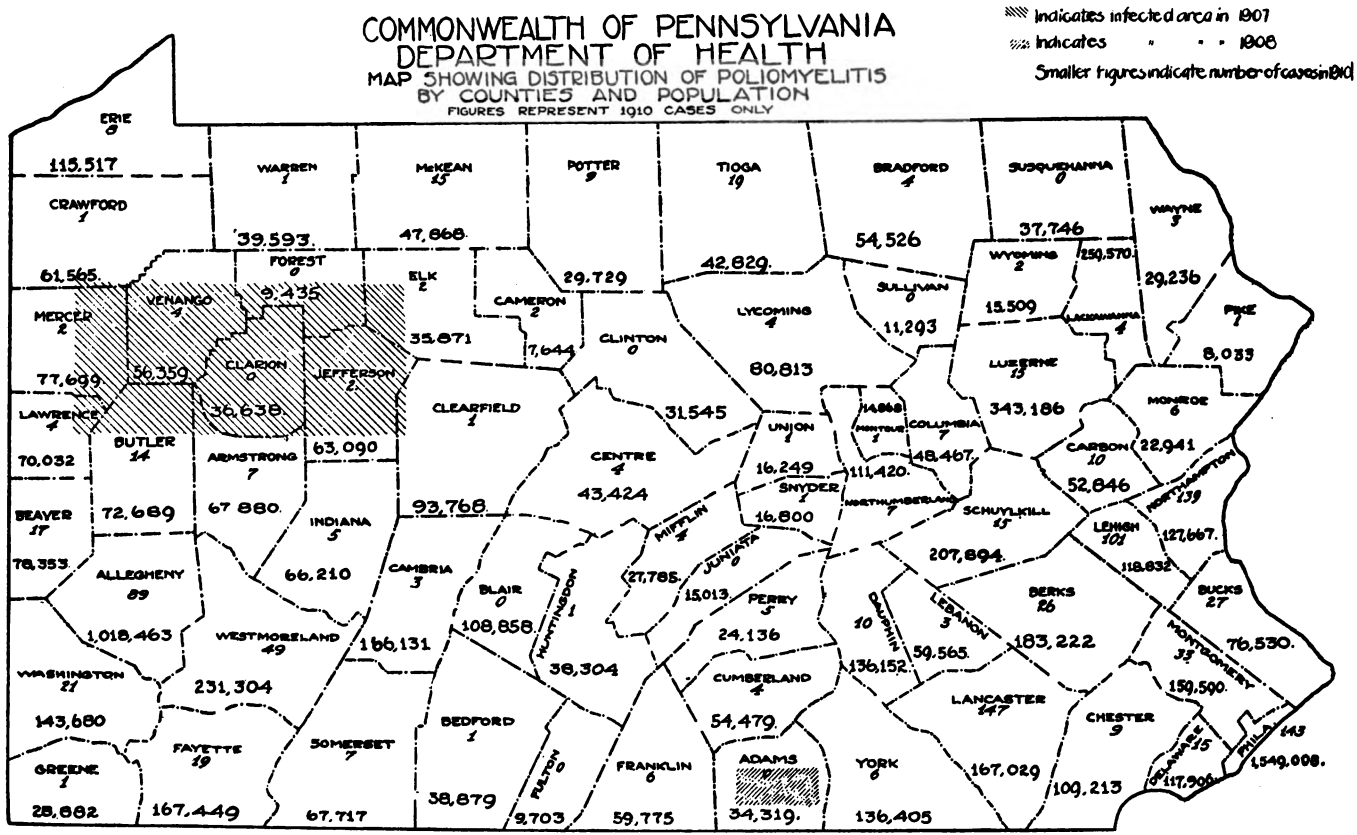

Fig. 4.—Map showing distribution of the disease, and also the population of the counties. The area indicated by broken black lines shows the district in northwestern Pennsylvania affected in 1907 and the district in Adams county affected in 1908.

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when grouped by day of disease on which count was made, is worthy of reproduction.

Day	Polynuclears	Small Lymphocytes	Large Lymphocytes	Eosinophils
2nd to 5th	52.2	34.2	10.1	3.5
6th to 9th	47.2	34.6	13.2	5.
After 10th	51.4	32.8	13.1	2.7

Ordinary leukocyte counts were made in a series of cases. The results seem to show a high degree of leukocytosis up to the ninth day of illness. Spinal fluids were studied eleven times, in each instance fluid being secured during the active stage of the disease. Neutral broth cultures showed the common diplococcus in every such fluid, but in no instance were we able to secure paralysis by inoculation with this organism.

A very large number of swabbings from the nasopharynx were secured and from fourteen such swabbings attempts were made to inoculate five monkeys by applying the centrifugal sediment resulting from macerating these materials in salt solution, swabbing the sediment over the mucous membranes and as nearly as possible placing sediment over the cribriform ethmoidal plate. All of these results were negative.

Six monkeys and seven rabbits injected under the skin with this material gave negative results. These findings seem to show that there is not great danger from the discharges, even though we do succeed in producing the disease by using the actual mucous membranes in the nasopharyngeal area.

Inoculation experiments made in an attempt to produce immunity in monkeys was to a very high degree successful. Attempts to give the disease to rabbits was only in part successful, as we failed absolutely in our attempts to transmit the disease from the rabbit developing paralysis to other rabbits or back to monkeys. It is possible that failure in this experiment occurred because the paralysis in the rabbit was not demonstrable until two weeks after inoculation.

In several communities an unusual number of yellow butterflies were reported. A series of these butterflies were collected, killed and macerated. Inoculation experiments from the resulting material failed uniformly to produce paralysis.

In the Bethlehems a blight among Norway maple trees attracted considerable attention and it was thought by many citizens that this disease was more prevalent near houses where cases of poliomyelitis

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TABLE 4.—SHOWING DETAILS OF CONTACT CASES

No.	Name	Contact	Manner	Same House	Date of Contact	Onset of Paralysis
160	J. Z.	From Trenton, N. J., direct contact in Lancaster on July 19. Indirect contact through father July 20	Direct and Indirect	No	July 19-20	July 20
20	H. H.	E. V. Philadelphia	Direct	No	3 yrs. or less	July 1
733	L. H.	D. F.	Direct	No	July 14	July 23
315	C. S.	N. D.	Direct	No	Aug. 24	Sept. 1
188	J. C. S.	H. G., New Brighton	Direct	No	Aug. 21	Sept. 1
1	M. M.	M. M.	Direct	No	July 27	Aug. 10
34	C. D.	K. L.	Direct	No	Aug. 26	Sept. 9
17	M. C.	L. T.	Direct	No	July 10	Aug. 24
715	A. E.	G. E.	Direct	Yes	Sept. 1	Sept. 10
499	F. O.	Case in same house	Direct	Yes	Aug. 5	Aug. 25
319	W. R.	Brother	Direct	Yes	Unstated	Aug. 9
577	C. H.	Brother	Direct	Yes	Aug. 25	Sept. 3
296	J. N.	Several cases in Scranton	Direct	No	July 3	July 10
492	W. G.	1619 Chew St.	Direct	No	July 10	July 12
501	J. K.	Neighbor	Direct	No	July 12	July 26
255	B. W.	S. H.	Direct	No	Aug. 18	Aug. 25
390	L. E. L.	Child. Smock, R. F. D.	Direct	No	Oct. 19	Nov. 2
670	J. S.	Philadelphia (L. children)	Direct	No	June 21	July 1
700	N. Y.	A. Y.	Direct	Yes	Constant	Sept. 27
704	W. Y.	A. Y.	Direct	Yes	Constant	Sept. 30
691	F. C.	E. F.	Direct	Yes	Constant	Aug. 7
696	S. B.	K. B.	Direct	Yes	Oct. 8	Oct. 11
693	H. R.	H. R., N. Bethlehem	Direct	Unstated	June 20	July 7
847	F. S.	Other case in family	Direct	Yes	Unstated	July 25
846	R. S.	F. S.	Direct	Yes	July 25	July 29
446	M. U.	J. C. (Slept in same bed) No. 527	Direct	No	June 15	June 26
261	M. H.	W. H.	Direct	Yes	July 24	Aug. 1
268	L. M.	Case 1067	Direct	Yes	Constant	Aug. 15
239	G. C.	K. C.	Direct	Yes	Constant	Sept. 11

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610	L. H.	N. H.	Direct	Yes	Aug. 17	Aug. 24
94	A. R.	M. R.	Direct	Yes	Oct. 31	Nov. 4
234	M. O.	C. O. (Case 554)	Direct	Yes	Constant	Sept. 3
329	F. O.	C. O. (Case 554)	Direct	Yes	Constant from 8/23	Sept. 2
342	M. W.	R. W. (Case 338)	Direct	Yes	Constant	Sept. 19
666	W. A.	Brother (Case 665)	Direct	Yes	Aug. 24	Sept. 8
602	M. H.	Brothers and sisters	Direct	Yes	Aug. 1	Aug. 7-8
585	C. E.	Sister	Direct	Yes	Aug. 1	Aug. 8
434	D. G.	Child from Huttles, Pa.	Direct	Yes	July 15	Aug. 11
743	H. W.	While waitress served a baby short time before who died from disease	Direct	No	Unstated	Oct. 5
1040	P. S.	Brother	Direct	Yes	Aug. 2	Aug. 9
2	M. M.	Visit of mother to other case	Indirect	No	Sept. 24	Oct. 13
393	P. C.	Grandmother from Lancaster ten days prior former contact with case	Indirect	No	Sept. 21	Oct. 1
606	— D.	Mother ironed Huber family wash. Case 602	Indirect	No	Aug. 1-8 inc.	Aug. 12
269	B. T.	Cases in neighborhood	Indirect	No	Unstated	Sept. 15
231	A. H.	Cases within a mile	Indirect	No	Unstated	Aug. 1
201	N. K.	Came from Trenton week previous to June 5	Indirect	No	Unstated	July 7
27	D. A.	Other case in neighborhood	Indirect	No	Unstated	Aug. 5
234	N. H.	Visited Lancaster Co.	Indirect	No	Sept. 25	Oct. 1
109	A. S.	Colerain Twp. Lancaster Co.	Indirect	No	About Aug. 1	Aug. 18
304	J. D.	In Pottstown prior to Sept. 24. In Elizabeth from Sept. 24 to 26	Indirect	No	Prior to Sept. 26	Oct. 1
657	J. C.	Allentown on or about Sept. 20	Indirect	No	Unstated	Oct. 1
325	C. W.	B. family	Indirect	No	Unstated	Sept. 20
357	F. T.	R. M. milk from M. cow	Indirect	No	Constant and Unstated	Oct. 14
385	E. Y.	M. case	Indirect	No	Unstated	Nov. 4
184	M. B.	Philadelphia	Indirect	No	Unknown	Aug. 8
363	B. S.	J. S. Brookville, R. D	Unstated	No	Aug. 1	Dec. 1
403	M. B.	S. V. Snedekerville, Pa.	Unstated	No	Sept.	Oct. 1

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existed. A careful examination of these conditions showed that the blight was not confined to the Norway maple and that there was no greater involvement of trees in the vicinity of the affected houses than elsewhere. Examination of the blighted branches showed a delicate mycelium running through the mesophyl of the leaves that was not identical with the oidium of Westphalia. The study of this mycelium gave us negative results. A large number of specimens of blood smears, samples of feces, urine and nose and throat swabbings were sent us by practitioners, but aside from finding microorganisms in the blood with great uniformity, nothing worthy of note was seen.

EXTRACT FROM LABORATORY REPORT PUBLISHED MARCH 2, 1911

In examining the blood from acute cases of poliomyelitis in human beings, and also in monkeys in which the disease was produced experimentally, an organism was found, different in morphologic characteristics from any heretofore described which may or may not, on further investigation, prove to be the etiologic factor in the causation of the disease. Blood smears being fixed in methyl alcohol for one minute and stained with carbol-thionin, the organism appears as a faintly-stained blue rod with regular cell wall about 10 microns long and about 8 microns in width, curved at an angle of 60 to 75 degrees at one end, occasionally at both ends. At times, the curved end is bulbous. Some of the organisms appear to have a very finely granular protoplasm when the highest amplification is employed. They may be discerned by means of a 4 mm. dry objective but their characteristics are much more satisfactorily delineated under the 1/12 oil-immersion lens. They are found free in the serum as well as within the body of the red blood-cell.

The organisms do not retain the violet color when stained by the method of Gram but assume the color of the counter stain which, as generally used in this laboratory, is a very dilute solution of carbol-fuchsin.

The bloods examined were from ten different cases of acute poliomyelitis in children and were taken during the epidemic of last summer and autumn, and from thirteen cases of the disease during the acute stage, which had been produced experimentally in as many monkeys.

Blood smears from three normal human beings were carefully examined and although the search for these organisms was diligently made, none was found. Smears were made from the bloods of thirteen normal monkeys with negative results. After inoculation with the virus these same monkeys gave positive results. The blood of other normal monkeys gave negative results.

Blood smears were stained with iodin and sulphuric acid in order to test the organisms for cellulose, but no blue stained organisms were seen.

Smears from the cords and brains of paralyzed monkeys, and from one human case were examined, but none of the new organisms was found.

Filtered virus stained with carbol-thionin and by Gram's method showed none of these organisms.

Defibrinated blood, three weeks to two months old, from two paralyzed monkeys, showed the forms in increased numbers.

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Cultures made from the blood of a paralyzed monkey, in blood bouillon, plain bouillon, and blood-agar, examined after having been inoculated three weeks, showed the presence of the organism in increased numbers. Dorsett's egg medium was inoculated with the same blood at the same time but the organism was not found in smears from the surface of the medium or from the water of condensation.

SUBSEQUENT FINDINGS

Since publishing the above preliminary report on the finding of rod-shaped microorganisms in specimens of stained blood taken from human beings suffering from poliomyelitis and from monkeys in which the disease had been produced experimentally, these microorganisms have been found moving freely in the serum or attached to the side of a red blood-corpuscle in fresh blood taken from three monkeys ill of poliomyelitis. The monkey blood was examined at the time of inoculation and at twenty-four-hour periods up to 168 hours after inoculation. In the blood examined at the time of inoculation and at the twenty-four-hour period no organisms were found. At the end of forty-eight hours, however, a few of the moving microorganisms were found. Their numbers in the fresh blood specimens were increased up to 120 hours, after which time they seemed to appear in about the same numbers till examination at regular intervals was discontinued. At the end of three weeks the motile organisms could still be found in large numbers in the fresh blood.

Examination of the blood of a normal monkey examined at the same period as a control, failed to reveal any of the microorganisms.

The motility was of a slow, sinuous type resembling that of a spirillum. The bulbous ends were readily discernible. At times the organism appeared as standing on end, presenting the bulb uppermost, so that it resembled a large coccus in the microscopic field. Presently it would right itself and the long curved rod terminating in this bulb could be distinctly followed.

Pure cultures of this organism were isolated from the bloods of these three monkeys, on a medium made of the central nervous system of a monkey, combined with agar-agar. After a generation or two on this central-nervous-system-agar, the organism grows well on the ordinary laboratory media.

CHARACTERS OF THE ORGANISM

The morphologic, biologic and biochemic characters of the organism grown on artificial media are as follows:

The best stain to bring out the morphology is carbol-thionin, though Loeffler's alkaline methylene-blue, Giemsa, Wright's, Ziehl-Nielson, carbol-

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fuchsin and Gram's method demonstrate the organism with no difficulty whatsoever. Stained with carbol-thionin the organism appears as a granulated rod, the young forms measuring 0.6 to 0.8 microns in width by 1 to 2 microns in length. The older forms may appear as long as 10 or 12 microns with a width of 0.6 to 0.8 microns. Many of the indeterminate forms appear as spirals with from two to four loose turns. The longer forms have in many instances bulbous ends which have somewhat the appearance of spores, though free spores have never been observed.

The organism is actively motile, having a single polar flagellum at one pole only. It has no demonstrable capsule, and it is decolorized by the method of Gram. Colonies on agar are circular, moist, convex, entire, 1 to 3 mm. in diameter. In color they are bluish gray by direct light, old colonies having a yellowish tinge by transmitted light. Their peripheries are translucent while their centers are thickened and appear yellowish-white. Microscopically they are brownish-white, with a finely granular translucent periphery, and an opaque, brown, grumous center.

Agar Slant: Bluish gray with a suggestion of greenish tinge in cultures three or four days old, moist, raised, regular edges, growth along whole length of streak. White beaded growth in stab.

Gelatin: White, beaded growth along needle track with nail-head, moist growth on surface. No liquefaction.

Potato: Moist, cream to brown, abundant.

Central-Nervous-System-Agar: Gray, moist, very abundant along entire path of inoculation.

Egg Medium (consisting of yolk only): Moist, very abundant, spreading, pinkish growth.

Bouillon: Very turbid, white flocculent sediment. In 120 hours a white ring around the glass tube at the level of the surface of the medium.

Litmus Milk: An initial acidity followed by a gradually increasing alkalinity, with a beginning reduction of the litmus at the bottom of the tube. No coagulation of the casein occurs. In four weeks acidification again takes place; reduction of litmus and firm coagulation after five weeks.

There is a slight production of acid in dextrose-agar and saccharose-agar, but not in mannite, maltose or lactose. Gas production does not occur in any of the sugars above mentioned. Mannite turns green after three weeks. The organism grows best in neutral medium, next best in acid, and poorest in alkaline.

Indol is not produced. It seems to grow most abundantly on media rich in phosphates. It does not grow well on media containing much

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TABLE 5.—SHOWING TRANSMISSIBILITY FROM HUMAN CASE TO MONKEY AND FROM MONKEY TO MONKEY

Virus, Case 53, Injected into

M. XXII Death from paralysis

M. XIX. Death from paralysis

M. XX. Paralysis without death. Legs remain paralyzed.

M. XXVII. Injected with filtered virus. Death from paralysis.

M. XIV b. Injected with virus. Death from paralysis.

M. XXIVa. Inj. with virus. Paralysis and death.

M. XIIb. Inj. with virus. Death from paralysis.

XXXIIa. Paralysis and death.

M. XXXVIII. Inj. virus. Merely depression. No paralysis.

M. XLI. Inj. with virus. Merely depression. No paralysis.

M. XXIX. Inj. filtered virus. Paralysis almost complete; recovery.

M. XXX. Inj. filtered virus. Paralysis followed by death.

M. XXXI. Inj. virus. Paralysis and death.

M. XXXV. Inj. with virus. Paralysis and death.

Filtrate of cords of human Case 53

Combined with Monkeys XXX, XXXII, XXXVIII, XLI and XXXV

M. XLIV. Paralysis and death.

M. XLVI. Inj. with virus. Paralysis and death.

M. XLIX. Inj. with filtered virus. Paralysis and death.

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glycerin. The organism grows in the absence of air as well as when the air is given free access.

EXPERIMENTAL PRODUCTION AND PREVENTION OF THE DISEASE

Experiments on the production of poliomyelitis in monkeys by using this organism are in progress.

We have published in connection with our laboratory work negative experiments resulting from a good many observations made with great care. On finding virus negative after an autopsy had been performed on a child dead with this disease, we conducted experiments to determine what agent in the embalming fluid might have been destructive, and, determining that formaldehyd and arsenic were both used in these fluids, a series of experiments were carried out with each, as well as with the fluid itself, solutions being injected at varying intervals as inoculation experiments to determine whether or not it would be possible to prevent the development of the disease by administration of these chemicals. The results in each instance were negative. Salvarsan was tried in a series of experiments in order that we might have data concerning the most potent arsenical preparation. These experiments, too, were negative, although they led to the incidental discovery of a positive Wassermann reaction in a monkey.

Abstract Case No. 256 (Laboratory Case No. 53).—G. T., female, aged 7; physician, Dr. George E. Day, Strasburg. Child well to August 23; August 24, stiffness of knee, pain in neck, fever, constipation, dizziness, temperature 102.5 F., no paralysis; August 26, paralysis right leg, both arms, loss of reflex, sensation normal; August 27, worse, pulse rapid, pressure symptoms; lumbar puncture secured 32 c.c. spinal fluid under excess pressure; August 28, slight improvement; 29th, breathing labored, 30th, death 9:00 a. m.; complete autopsy 11:00 a. m. Abundant material for laboratory study was obtained and true pathologic picture found. Tissues gave Noguchi butyric reaction. Emulsion from lumbar cord, bulb, pons, lymph-nodes, cultures from cerebrum, cerebellum, pons, bulb, lumbar cord, lymph-nodes; Gram positive coccus found in cultures taken at autopsy from brain and cord; nothing further by dark-field illumination. Monkeys inoculated with this coccus remained well. Two monkeys received emulsion from cord, pons and bulb into brain; both contracted disease and from this strain of virus successful transmission experiments were secured as illustrated on protocol of transmissibility.

I am greatly indebted to Dr. B. Franklin Royer, Chief Medical Inspector, and the staff of the Division of Medical Inspection, for the compilation of the statistics in this report, and to Drs. Herbert Fox, and J. B. Rucker, Jr., of the Laboratory Division, for the notes of the experimental work carried out under my direction.

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INFANTILE PARALYSIS

WITH ESPECIAL REFERENCE TO ITS OCCURRENCE IN MASSACHUSETTS, 1907-1910 *

ROBERT W. LOVETT, M.D., AND MARK W. RICHARDSON, M.D.
BOSTON

The problem of infantile paralysis is definitely before the physicians of the United States.

In the twenty years from 1881 to 1900 there were reported in this country 200 cases. In the five-year period beginning in 1900 and ending in 1904 there were reported 100 cases. In the five years beginning in 1905 and ending in 1909, 5,400 cases, and in one — the year 1910 — approximately 9,000 cases. Such an extraordinary increase, in which Europe has in no way shared, brings before us very seriously our obligation to investigate the disease, to take account of our stock of knowledge regarding it and to do what we properly can do to quiet the panic that exists in the minds of the laity, by placing before them what we know and by asking their assistance to help us to learn more (Charts 1, 2 and 3).

The present paper will consist of a brief résumé of our present accepted knowledge of the various aspects of the disease and an analysis of the studies in Massachusetts made by the State Board of Health for the four years beginning in 1907.

*This paper was prepared with the co-operation of Henry P. Walcott, M.D., Chairman of the Massachusetts State Board of Health, and of Theobald Smith, M.D., Milton J. Rosenau, M.D., John Lovett Morse, M.D., and James H. Wright, M.D., Advisory Committee on Infantile Paralysis to the Massachusetts State Board of Health.

In the field work the Board has had for two years (1909-1910) the services of Dr. Philip A. E. Sheppard of Boston, and Dr. Thomas P. Hennelly, now of Pittsfield, Mass. The epidemic in the Deerfield Valley in 1908 was investigated by Dr. Herbert P. Emerson, of Springfield, then State Inspector of Health for that district. Much valuable assistance has been rendered furthermore by the other State Inspectors of Health throughout the Commonwealth.

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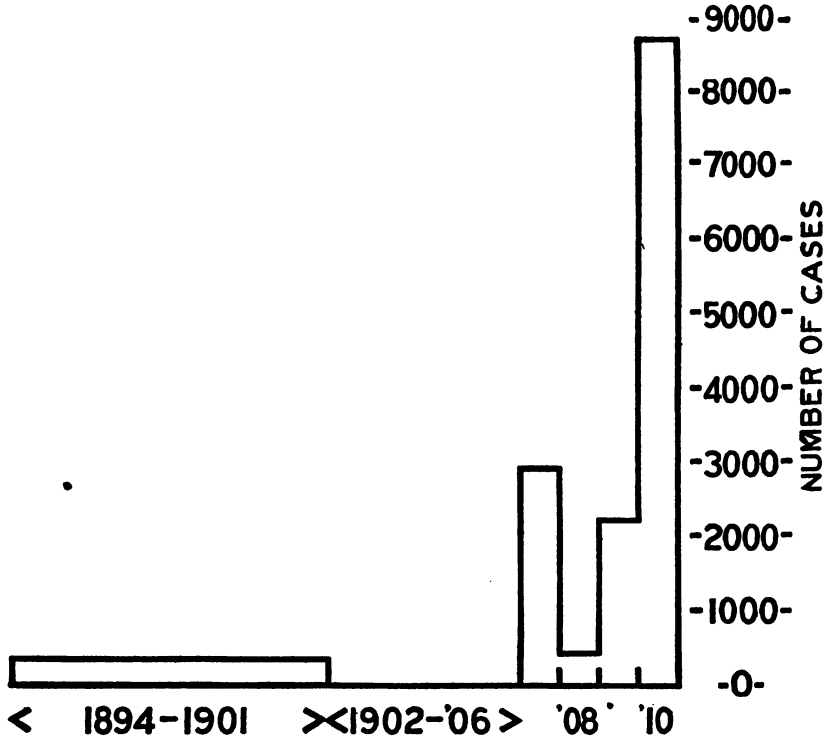

Chart 1.—Prevalence of infantile paralysis in the United States, 1894 to 1910.

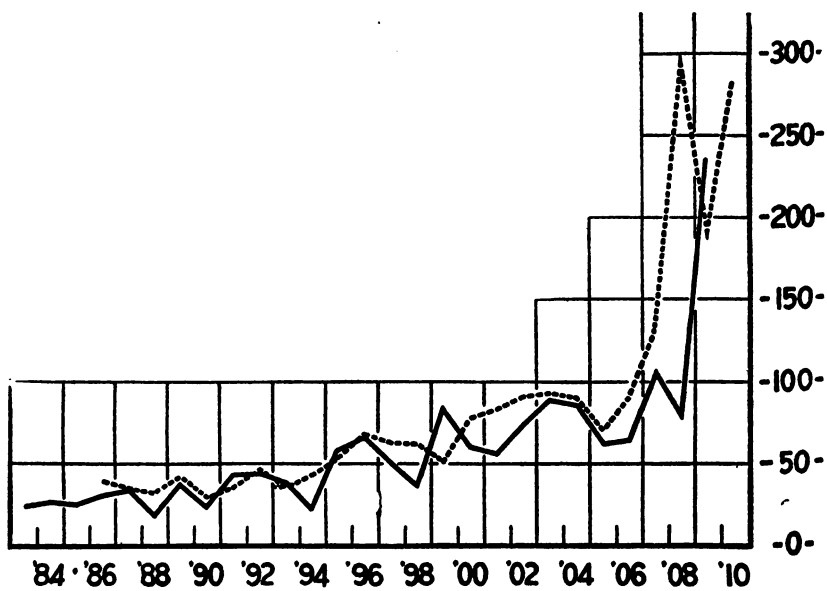

Chart 2.—Number of new patients with infantile paralysis treated at Children's Hospital, Boston, indicated by the solid black line; those treated at the New York Orthopedic Dispensary and Hospital by the dotted line; both by years.

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I. RECENT HISTORY AND ETIOLOGY OF INFANTILE PARALYSIS

RÉSUMÉ OF PRESENT ACCEPTED KNOWLEDGE

History.—The disease is not a new one. Although the first clear description of it was given in 1840 by Heine, a German, it was fairly well described by an English physician, Michael Underwood, in 1774. Since the perfectly clear description of the disease by Heine, in 1840, successive additions to our knowledge have been made, the most impor-

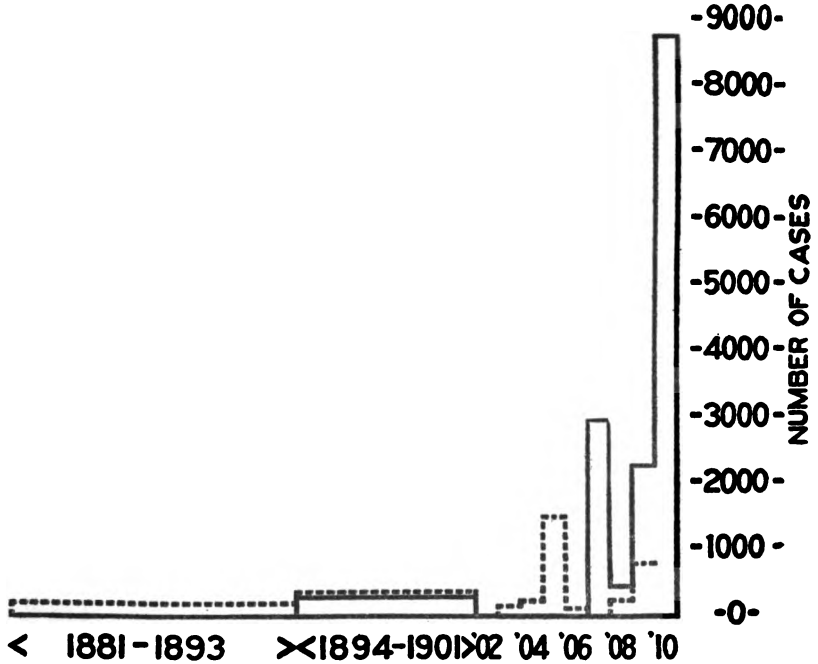

Chart 3.—Relative prevalence of infantile paralysis in the United States and Europe and Australia; the solid black line refers to the United States; the dotted line to the following countries: Italy, Sweden, Norway, Germany, Austria, France, England and Australia.

tant contributions until very recently having been by Norwegian and Swedish authors, who had unusual opportunities for the study of the disease between 1899 and 1907.

Infectious Character.—Although it had long been suspected to be an infectious disease, this fact was not absolutely proved until November, 1909, when Flexner and Lewis of the Rockefeller Institute in New York

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demonstrated, by inoculation of a series of monkeys with tissues from children dying of the disease, that the process was an infectious one, placing it in the category with rabies, yellow fever and foot-and-mouth disease. This discovery was undoubtedly the most important addition to our knowledge of the disease ever made.

Epidemic Character.—That the disease was infectious was suggested by the fact, recognized as early as 1881, that it occurred in epidemics. Indeed, a questionable epidemic was reported in 1841 by a Louisiana physician. From 1881 until the present time, epidemics have been reported with increasing frequency, and since 1905 the size of epidemics has largely increased. We must remember, however, that of late greater attention has been paid to the disease and a larger number of cases has been recognized, so that the increase is in a measure apparent. Nevertheless, the increase has been too great to be accounted for by any theory of increased attention to the disease.

Epidemic History.—The first great epidemic of modern times occurred in Norway and Sweden in 1905, 2,000 cases being reported in Norway and Sweden together. In 1907 the first great American epidemic occurred, 2,500 cases being reported in and about New York, since which time the disease has been increasing irregularly in the United States, making a very great increase in 1910 over any previous record.

Infantile paralysis first appeared in the United States to any considerable extent in 1894, when Caverly of Rutland reported 132 cases in the Otter Creek Valley, Vermont. Some ten small epidemics were reported in the United States during the next twelve years, and in 1907 the largest epidemic on record, that in New York, occurred, some 2,900 cases having been reported in that year. In 1908 the disease was apparently quiescent, only 392 cases being reported in the United States. This was followed by outbreaks in 1909 in various parts of the country, and in Cuba for the first time, with a total for the year of 2,343 cases in all.

In 1910 the disease assumed much more serious proportions and was present in forty-three states. Several states had epidemics, 500 or more cases occurring in the District of Columbia, Iowa, Massachusetts, Minnesota, Indiana and Pennsylvania, and from 200 to 500 in Kansas, Maryland, New Hampshire, New York, Rhode Island, Virginia, Washington and Wisconsin (Chart 4).

The reasons for this marked increase in the occurrence of infantile paralysis are not clear, but certain considerations are of interest and possibly of importance.

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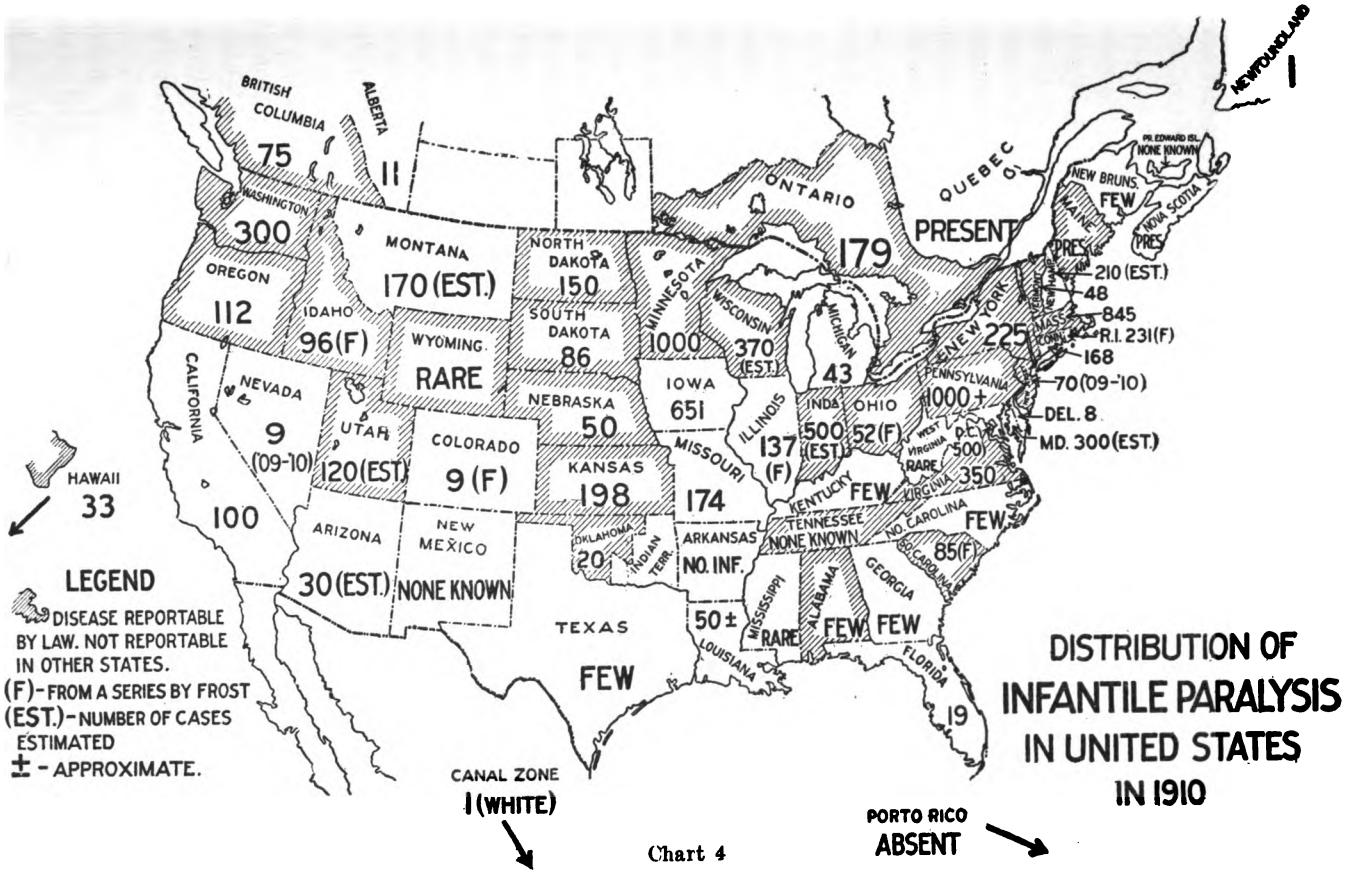

DISTRIBUTION OF INFANTILE PARALYSIS IN UNITED STATES IN 1910

Chart 4

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Question of Importation.—No explanation has been given for the sudden increase of the disease in Norway and Sweden in 1905, but after that epidemic it seems not unreasonable to suppose that the disease may have been increased in this country by Scandinavian immigrants, who arrived to the number of about 42,000 in the year 1907. The epidemic of 1907 in New York may have had its origin in this way, and the disease may have been carried in a similar manner to the middle west, but the explanation is not wholly satisfactory, inasmuch as the outbreak of the disease in the west did not conform closely to the distribution of Scandinavian immigrants. The Massachusetts epidemic can be reasonably explained on the ground that it came from New York. In fact, the New York epidemic was sufficiently large to be a possible source for the outbreaks in various parts of this country.

Travel.—It has been noted in the distribution of all epidemics that they tend to spread along the lines of greatest travel; certain figures were, therefore, analyzed with regard to passenger traffic in the United States.

Steam Railways.—In 1904 there were 715,400,000 passengers carried by the railroads, and in 1908 there were 890,000,000, the average number of passengers per train mile being in the first period forty-six, and in the second fifty-four; that is, there were 175,000,000 more passengers carried per year in 1908 than in 1904.

Electric Railways.—The only available figures with regard to electric railway traffic are those for 1902 and 1907. In 1902 the number of passengers carried was 5,536,000,000, and in 1907 it was 9,500,000,000, an increase in round numbers of 4,000,000,000, or about 75 per cent. in five years.

Automobiles.—Another important factor as regards increased travel is the automobile, and here the only figures which it has been possible to analyze are those relating to Massachusetts. In 1904 there were 4,000 automobiles registered in Massachusetts; in 1909, 24,000; that is, in the five-year period the number of automobiles increased six times.

Conclusions from Study of Travel.—These figures show a very decided increase in travel in the United States in five years, an increase wholly out of proportion to the increase in population. It is not at all unlikely that it is one factor by which the disease has been distributed over the country; and if this conclusion is applied to the year 1894 when the Rutland epidemic occurred, the increased communication by travel becomes very much more marked by comparison.

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Having considered these general aspects of the disease we now come to the closer consideration of its etiology.

ETIOLOGY

The disease is an infectious one, caused by an ultramicroscopic virus which passes through the closest porcelain filters, its infectious character having been proved by the fact that it can be propagated in a second and many subsequent series of monkeys from tissues of a first series.

The virus is contained in the brain and spinal cord, the mucous membrane of the nasopharynx, in infected lymph-nodes, in the salivary glands, and, in the acute stage, in the cerebrospinal fluid and the blood.

The disease may be produced experimentally in monkeys by intracerebral, intraperitoneal, subdural, intraneural, perineural and subcutaneous injections, and by implantation in the anterior chamber of the eye. The disease has been caused also by the introduction of the virus into the stomach and intestines. Successful inoculation has been accomplished by rubbing the virus into a scarified or even a sound nasal membrane, and two observers succeeded in producing the disease by inhalation of an emulsion containing the virus and by implantation in the trachea. All investigators agree, however, that the intracranial route is the best for inoculation.

Inoculation of horses, calves, goats, pigs, doves, sheep, rats, cats, mice, chickens, guinea-pigs and dogs have proved negative, and monkeys have proved the most satisfactory animals for study. A paralysis may be experimentally produced in certain varieties of young rabbits and may be transmitted from one generation to another of rabbits and finally back to monkeys by successive inoculations.

The virulence of a suspension is not impaired by drying (to nine days), by freezing, nor by suspension in glycerin for five months, but it is killed by exposure to a temperature of from 45 to 50 C. in half an hour. It is also killed by such weak disinfectants as 1 per cent. solution of peroxid of hydrogen (perhydrol of Merck) by 1 to 500 solution of permanganate of potash, and by a powder containing menthol 0.5, salol 5, and boric acid 20.

The organism is not visible with the highest powers of the microscope and will not grow on the culture media in general use.

The disease in monkeys is very much like that in children, but is much more fatal; in fact, so large a proportion of inoculated monkeys

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die that the study of the affection clinically in them is difficult. No instances as yet have been reported in which one monkey has taken the disease from another, although long continued and intimate contact has been maintained.

The incubation period in monkeys is from five to forty-six days, with an average of eight or nine days.

IMMUNITY

A child or animal that has once had the disease becomes apparently immune and the blood of children and monkeys recovered from the disease has been found to render other monkeys refractory to subsequent inoculation, the specific antibodies persisting in the blood for three years in one case, but being absent in another case eleven years after the onset.[1] This fact, of course, suggests that a preventive serum may be hoped for, but as yet the serum treatment is wholly in an experimental stage, and not yet ready for use in cases of the human disease.

PERSONAL TRANSMISSION — DIRECT AND INDIRECT

In discussing the question of the transmission from patient to patient, the use of the technical and restricted term "contagious" will be avoided and the term "transmissible" will be employed.

The disease may be transmitted apparently in three ways: (1) By direct contact of a healthy child with a sick one; (2) by indirect contact by means of healthy persons passing from the sick to the well; and (3) in certain very rare instances a child has become ill shortly after occupying a house from which an affected family had moved.

Comparative Transmissibility.—The disease is, however, apparently less transmissible than other so-called contagious diseases with which we are more familiar.

In Minnesota a very careful investigation was conducted by Hill as to the relative transmissibility of infantile paralysis compared with that of scarlet fever, measles, etc., in the same territory. Of persons known to have been exposed to diphtheria, scarlet fever and infantile paralysis, the percentage contracting the disease was as follows: Scarlet fever, 22 per cent.; diphtheria, 17 per cent.; infantile paralysis, 6 per cent. Taking the instances of these same diseases, where one case existed in a family, the number of other cases occurring in the same family was as

1. Netter and Levaditi: Comptes rend. Soc. de biol., 1910, lxviii, 855.

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follows: Scarlet fever, 40 per cent.; typhoid fever, 30 per cent.; diphtheria, 29 per cent.; infantile paralysis, 17 per cent. The disease is thus apparently less transmissible than scarlet fever, typhoid fever or diphtheria.

The same evidences of mild transmissibility were shown in an investigation by Emerson, of an epidemic in the Deerfield Valley in Massachusetts in 1908, where a rural community offered excellent opportunities for study of contact. Furthermore, such instances as the following are difficult to explain by any theory of personal contact, direct or indirect:

In a sporting camp in a northern forest the 5-year old son of the proprietor had been in the camp since early spring. He had never left the camp during the summer. The camp was 5 miles from any other camp or house and 10 miles from the frontier town. There had been no other children in the camp during the summer and no illness of any kind in the guests or among animals. The few guests who were there came from the large cities, put on their hunting and fishing clothes and lived out of doors. In October the child developed a typical infantile paralysis, which still persists in one leg.

Evidence of transmission of the disease by personal contact from other epidemics is to be found in Wickman's Study, Shidler's report,[2] an epidemic observed by Frost,[3] and the Massachusetts studies.

Possible Factors Other Than Personal Transmission.—We come next to the consideration of the possibility of fruit, dust, insects, and animal paralysis as factors in the causation of infantile paralysis.

Fruit.—As to fruit, the high cost of living has undoubtedly led to a diminished consumption of meat, and presumably to an increased consumption of fruit. Traffic in fruit has increased steadily between 1900 and 1908, the increase being from one to two million tons a year, up to 1905, since which time there has been no such sharp increase. So far as these figures go, therefore, it would not seem that any very striking factor was to be found in this analysis. Common observation would, however, seem to show that in Massachusetts, at least, there are more fruit stores and apparently more fruit in use than was the case five years ago.

Dust.—In order to determine the rôle of dust in the etiology of infantile paralysis, the rainfall of the country was analyzed for two

2. Shidler: Pediatrics, 1910, xxii, 539.
3. Frost: Public Health Bulletin 44.

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periods: first, the five years from 1900 to 1904; and second, the five-year period from 1905 to 1909, when we had the greater prevalence of the disease. The total rainfall during the last five years on the Atlantic seaboard has shown everywhere a diminution, reaching a high figure in New England. On the contrary, the regions in the middle west affected by the epidemic of infantile paralysis in 1909 showed an increased rainfall during the last five years, the rainfall being as much in excess of the normal as in New England it was deficient. In the middle Atlantic states, however, including New York, the rainfall in the two periods was practically the same. Excessive dryness in the last five-year period cannot, therefore, be put down as a condition pertaining to the affected regions, if one considers New England along with Nebraska and Minnesota. But we must remember that the germ is not injured by freezing or drying, and that the season of occurrence is certainly the season of greatest occurrence of dust. Moreover, a German observer found the disease in an unduly large proportion of shoemakers' children who were much in the shops where the dried dust was flying about, and the distribution of the disease along lines of travel suggests the possibility of such a mode of transmission.

Insects.—There are certain facts connected with the disease which suggest the possibility of its being transmitted by insects. This matter will be dealt with in speaking of the Massachusetts studies.

It has been shown experimentally by Flexner that if flies are allowed to feed on the cord of a monkey dying of the disease that they may carry the virus for forty-eight hours.[4]

Domestic Animals.—With regard to paralysis in the domestic animals, cases of paralysis strikingly suggestive are reported in horses, pigs, cats, dogs, rabbits, chickens, and other animals. A study of the distribution of animal paralysis in Massachusetts by means of letters addressed to veterinarians and animal inspectors showed, however, that in 1909 the distribution of cases of animal paralysis so reported did not in any way correspond to the distribution of the human disease for the same year, but much more extensive investigation along this line is necessary.

The conclusion must be that, in a consideration of etiology, infantile paralysis cannot be dismissed as a disease due only to transmission from patient to patient, but that these other factors must also be considered as possibly important.

4. Flexner, Simon and Clark, Paul F.: Jour. Am. Med. Assn., June 10, 1911, p. 1717.

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Season of Occurrence.—In considering the etiology of the disease one is struck with the remarkably uniform seasonal occurrence which prevails in the north or in the tropics, in Europe or America. The disease begins to appear in the early summer, reaches its height generally in July, August or September, and diminishes through October, November and December, nearly to disappear in late winter and spring. In the southern hemisphere this behavior is reversed and the disease prevails most there in February and March, which is, of course, late summer and early fall in that locality.

Age.—The age of selection of the disease is between 2 and 3. Adult cases are comparatively uncommon, and occur chiefly in young adults. Dividing the ages into five-year periods, the following table was constructed by Frost:

TABLE 1.—AGE INCIDENCE IN FIVE-YEAR PERIODS EXPRESSED IN PERCENTAGES

Age Years	N. Y. Commission (729 Cases) Per cent. of Total	Lovett (Mass.) (615 Cases) Per cent of Total	Hill (Minn.) (325 Cases) Per cent. of Total
Under 1	8.5	7.2	6.5
1 to 5	82.0	64.5	48.6
6 to 10	6.4	15.9	23.7
11 to 15	1.9	5.0	7.7
16 to 20	0.68	2.4	6.5
Over 20	0.40	5.0	7.0

General Incidence.—It affects all classes, rich and poor, in the cities and the country, in the tenements and in detached houses, on the top floor of houses, on the ground floor and in basements.

II. PATHOLOGY, SYMPTOMS, DIAGNOSIS, PROGNOSIS, PREVENTION AND TREATMENT

General Grouping of Symptoms and Types.—As a working hypothesis we may keep in mind the following as to symptoms and types of paralysis:

The infection enters the body by channels as yet unknown. In some instances this infective agent causes death from general paralysis due to extensive destruction of certain spinal and cerebral centers; in a second class of cases this agent causes some permanent loss of power in part or all of one or more limbs, the back, the abdomen, or in groups of muscles supplied by cranial nerves. In a third class, the same agent causes a very local yet probably permanent paralysis in one small muscle

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or small group of muscles, e. g., the extensor muscles of one little finger or certain muscles of the spine.

In a fourth class, weakness or transitory paralysis ending in recovery occurs as the result of the same infection. And lastly, in a fifth class, the infective agent causes fever, prostration and perhaps pain, but does not cause sufficient destruction to bring about any paralysis or weakness.

If we recognize this descending scale in the severity of the disease as depending probably either on the dose of the poison received or on the resistance of the individual, and recognize that the involvement of the spinal centers is perhaps after all incidental rather than essential, we may well believe that the disease is far more prevalent than is generally supposed, and especially in its abortive forms.

PATHOLOGIC ANATOMY AND HISTOLOGY

The characteristic lesions of the disease are found in the spinal cord and are most marked in the anterior horns of the gray matter of the cervical and lumbar enlargements.

In the gray matter in the early stages of the lesions are

1. Congestion of blood-vessels.

2. Edematous softening, sometimes accompanied by hemorrhage.

3. Infiltration with cells. (The cells diffusely infiltrate as well as aggregate together in foci and about the blood-vessels. They are of various kinds, but are chiefly lymphocytes and larger cells whose origin is not generally agreed on, and which are sometimes phagocytic.

4. Degeneration and diminution in the number of nerve-cells. The cytoplasm of the nerve-cell becomes hyaline and disappears leaving the nucleus naked and necrotic, and it also eventually disappears. The degenerated cell may be surrounded and invaded by the infiltrating cells. All of the nerve-cells in a given area do not always show degenerative changes, and more of them may be involved in one side of the cord than in the other. In the later stages of the disease, in extensively affected regions, nerve-cells and their processes are absent, the tissue is disintegrated and there is infiltration with phagocytic cells, plasma cells, proliferated glia cells, and connective tissue cells. Eventually, neuroglia with some connective tissue replaces the disorganized tissue and the part may appear atrophied to the naked eye.

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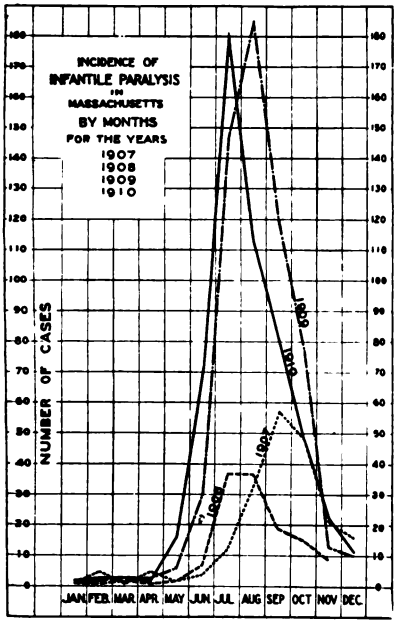
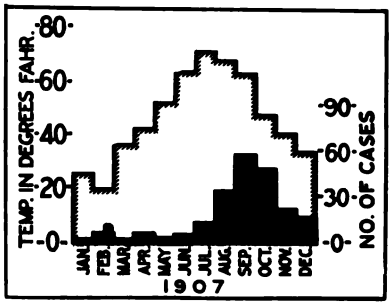


Chart 5.



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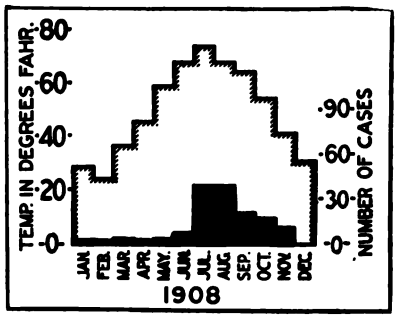

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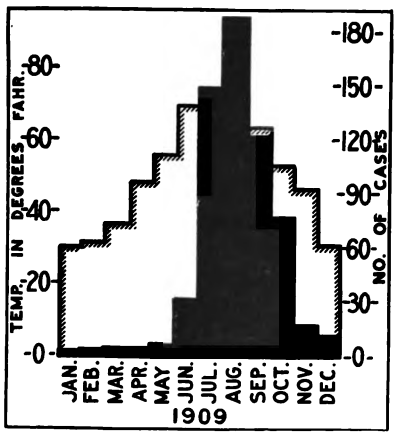

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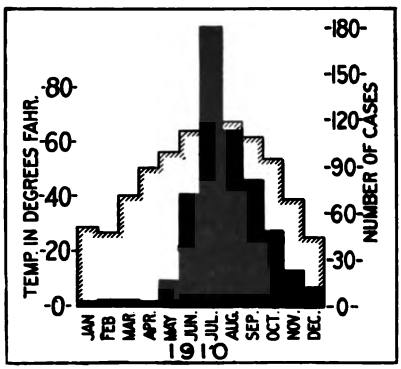

Chart 9

Chart 6.—Incidence of cases in 1907 by months shown in solid black; mean temperature in white.

Chart 7.—Incidence of cases in 1908 by months in solid black; mean temperature in white.

Chart 8.—Incidence of cases in 1909 by months in solid black; mean temperature in white.

Chart 9.—Incidence of cases in 1910 in solid black; mean temperature in white.

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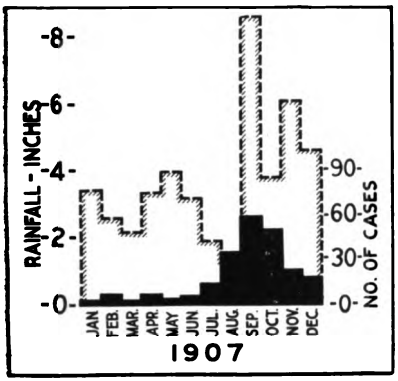

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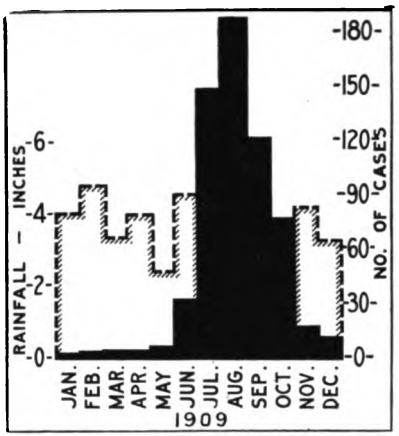

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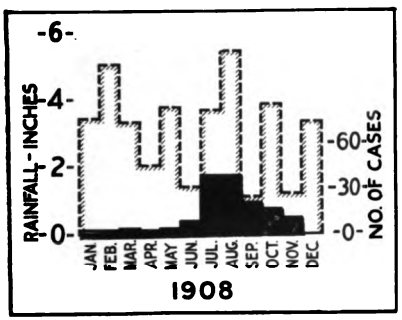

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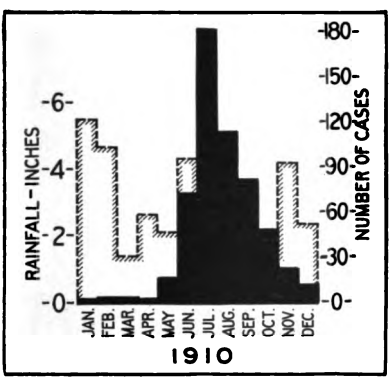

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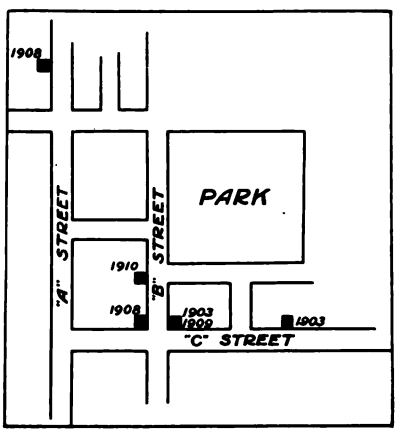

Chart 14

Chart 10.—Incidence of cases and rainfall by months in 1907. The former shown in solid black.

Chart 11.—Incidence of cases and rainfall in 1908. The former shown in solid black.

Chart 12.—Monthly incidence of cases and rainfall in 1909. The former in solid black.

Chart 13.—Monthly incidence of cases and rainfall in 1910. The former in solid black.

Chart 14.—Map showing endemic focus of infantile paralysis in a city of 119,000 inhabitants; cases occurring in the years 1903, 1908, 1909 and 1910.

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In the white matter in the early stages of the disease, the lesions consist of congestion, edema and cellular infiltration of the adventitial lymph spaces of the blood-vessels and to a slight extent of the white matter as well. The infiltrating cells are chiefly lymphocyte-like cells. Small foci of these cells have been noted in the white matter. In some cases, acute degenerative changes have been observd in the anterior roots.

The pia mater also shows some infiltration, chiefly with lymphocytes, which is greatest in the anterior portions of the cord in the lumbar and sacral regions.

In the spinal ganglia there may be the same infiltration as in the white matter, without marked changes in the ganglion cells.

In the later stages of the disease there is degeneration and atrophy of nerve fiber tracts, anterior roots, motor nerves and muscles, depending in extent and location on the nerve-cells which have been destroyed.

In the medulla and pons similar lesions to those in the cord are found, but the degeneration of the ganglion cells is not so extensive as in the cord.

The brain as a rule shows comparatively slight lesions. Congestion of blood-vessels, small hemorrhages, cellular infiltration of the vessel sheaths and of the brain substance in foci have been observed in various situations. In old cases, foci of sclerosis have been observed.

In acute cases, the thoracic and abdominal organs show changes which may be observed in other acute infectious diseases. A hyperplasia of the lymph-adenoid tissue of the intestine, mesentery and spleen is commonly observed in addition to acute degeneration of the myocardium, liver and kidneys, and there may be bronchopneumonia.

There is nothing in the pathologic anatomy and histology of the disease which shows the mode of entrance of the infectious agent into the body nor whether it reaches the central nervous system by the blood or by the lymph-stream.

SYMPTOMATOLOGY OF INFANTILE PARALYSIS

It is impossible in the time at our disposal to give more than the most cursory description of the symptomatology of this disease. If we think of it as a poliomyelo-encephalitis with meningeal complications and appreciate the fact that the disease may affect any or all parts of the gray matter of the central nervous system, and in all possible combinations, it is easier to understand the innumerable manifestations of the disease and it is less likely to be overlooked and mistaken for other conditions.

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The best classification of the various manifestations of the disease is that of Wickman, which is as follows:

1. Ordinary spinal paralysis; anterior poliomyelitis.
2. Progressive paralysis, usually ascending, less often descending; Landry's paralysis.
3. Bulbar paralysis; polio-encephalitis of pons.
4. Acute encephalitis; giving spastic mono- or hemiplegia.
5. Ataxic type.
6. Meningitic type.
7. Polyneuritic; multiple neuritic type.
8. Abortive type.

Initial Symptoms.—It will be well, perhaps, before taking up the special clinical types of this disease to consider briefly its initial symptoms and the possibilities of making a diagnosis before the appearance of the paralysis. There is, in the vast majority of cases, nothing characteristic about the onset. The symptoms are, in general, those of an acute infection. In many instances, however, gastro-intestinal symptoms predominate, while in others those referable to the respiratory tract are the most marked. Sweating, marked nervous irritability and general hyperesthesia are present in many instances before the onset of the paralysis, but they are not at all constant. Their presence points strongly toward this disease but their absence does not count against it. It is safe to say, therefore, that at present there is nothing about the early symptoms to justify a positive diagnosis of infantile paralysis before the onset of the paralysis, although the appearance of the symptoms of an acute infection in a child, known to have been exposed to this disease or during an epidemic, especially if accompanied by sweating, nervous irritability and general hyperesthesia, are sufficient to justify a probable diagnosis.

A leukopenia has been found in animals during the late prodromal and early part of the acute stage. It is, however, not a constant symptom in man and is often replaced by a hyperleukocytosis. It is possible that there is a lymphocytosis in the early stages, but the evidence at present is not sufficient to show whether it is at all constant. It is possible that both the absolute and differential count of the leukocytes may prove useful in the early diagnosis of infantile paralysis. At present, however, they are of little or no assistance.

Experimentally, there is an increase in the number of cells in the cerebrospinal fluid during the prodromal stage, the polynuclear cells

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at this stage outnumbering the mononuclear. After the appearance of the paralysis, however, the mononuclear cells quickly exceed the polynuclear. A fibrin clot is also often formed during the prodromal or early part of the acute stage. The cerebrospinal fluid, in the only reported instance in which it has been examined in man during the preparalytic stage, was slightly opalescent, gave a marked protein reaction with Noguchi's test and showed an excess of polynuclear cells. The formula had begun to change to the lymphocytic, however, before the appearance of the paralysis. It seems safe to say, therefore, that in most instances a positive diagnosis of infantile paralysis can be made in the preparalytic stage by means of lumbar puncture, and that this is the only way in which a positive diagnosis can be made at this time. The value of this procedure is considerably limited, however, by the variability of the early symptoms and the consequent lack of anything to definitely suggest its use. In order to recognize the disease early, therefore, before the onset of the paralysis, it would be necessary to do a lumbar puncture on every sick child, which hardly seems a rational procedure.

Abortive Type.—What has been said as to the symptomatology and diagnosis of the preparalytic stage of infantile paralysis applies equally well to the abortive type (Type 8 under Wickham's classification), meaning by this term the type in which there is never any paralysis. There has been some doubt as to the existence of this type in the past, because there was no way of positively proving that the cases supposed to be of this type really were examples of this disease at all, the evidence being merely presumptive. Flexner and Clark and Frost have, however, recently demonstrated the presence of specific immune bodies in the blood of persons supposed to have had abortive attacks of infantile paralysis. The blood serum of these patients inactivated the virus of the disease, so that the infection of monkeys was impossible. There is no doubt, therefore, that abortive cases do occur. It is probable that they are as common as, if not more common than, those with paralysis. The importance of the recognition of these cases, if the present conception as to the rôle of "carriers" in the spread of the disease is correct, is obvious.

1. Ordinary Spinal Type.—It hardly seems necessary to take up in detail the symptomatology of this type, with which everyone is familiar. The paralysis reaches its maximum in from a few hours to three or four days, is followed by a stationary period of from one to four weeks, which is in turn followed by a period of improvement, lasting from six months

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to a year, the final paralysis always being less than the initial. It is impossible to prophesy in the acute stage how extensive the final paralysis will be. In general, however, the final paralysis varies directly with the initial. The final paralysis is always flaccid; its distribution usually monoplegic or hemiplegic. In most instances some of the muscles of the paralyzed extremity escape; not infrequently only one or two are involved. The reflexes are diminished or absent, sensation is only exceptionally impaired. Deformities develop as the result of contractures of the non-paralyzed muscles.

It is not very uncommon for other muscles than those of the extremities to be involved, while these escape. Such muscles are most often the erectors of the spine and the abdominal muscles. The involvement is, moreover, not infrequently unilateral. The extensor muscles of the neck, those of one side of the spine or of one side of the abdomen may, for example, be alone involved. The deformities resulting from the paralysis or paresis of these muscles are often misunderstood and attributed to other causes.

2. Progressive Type.—In this type the paralysis usually appears first in the legs, and gradually extends upward. In rare instances it appears first in the arms, extends downward and finally upward to the muscles supplied by the medulla. It is probable that the great majority of the cases that have in the past been described under the term of "Landry's paralysis" really were examples of this type of infantile paralysis. When the paralysis reaches the external muscles of respiration, as it very often does, death is practically inevitable and usually occurs on the third or fourth day. The diaphragm is also sometimes involved. Death occurs sooner, of course, when this happens. This form must not be confused with that in which death results from respiratory paralysis due to involvement of the centers of respiration in the medulla.

3. The Bulbar Type.—Cases of this type have in the past been usually designated as polio-encephalitis superior or inferior, according to which of the cranial nerve nuclei were involved. The nuclei may be affected singly or in all possible combinations. The symptoms depend, of course, on the nuclei involved. The facial and abducens nerves are perhaps the most often affected. There may be paralysis of deglutition and of the muscles of the larynx. When the vagus is involved there are disturbances of respiration and of the cardiac action. In such instances, the respiration is often of the Cheyne-Stokes type and the prognosis is practically

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hopeless. Involvement of one or more of the cranial nerve nuclei is not very uncommon in connection with the ordinary spinal type of infantile paralysis. When this happens the resulting picture is a combination of the two types.

4. Acute Encephalitic Type.—This type was described by Strümpell many years ago under the term, "acute encephalitis of children," but it has only recently been recognized as a variety of the disease under consideration. The symptoms resemble those of acute meningitis, the deep reflexes are, as a rule, exaggerated and the paralysis is spastic. The diagnosis is usually impossible without lumbar puncture. The prognosis as to life is better than it would appear and that as to recovery from the paralysis much better than in paralysis due to other cerebral diseases.

5. The Ataxic Type.—Ataxia is a prominent symptom in a certain number of cases. In a few, it is the only nervous symptom; in others, it is associated with paralysis of the cranial nerves and sometimes with a small amount of spinal paralysis. The ataxia is often distinctly of the cerebellar type.

6. The Meningitic Type.—Symptoms of meningeal irritation are not at all uncommon in the early stags of all types of infantile paralysis. These are so marked in many instances that they present the typical picture of meningitis. Headache, rigidity of the neck and back, vomiting, tonic and clonic spasms, strabismus, Kernig's sign, delirium, coma and other signs of meningeal irritation may be present in any and all combinations. In such cases the diagnosis on the symptomatology alone is impossible before the appearance of the flaccid paralysis. Even then it is open to doubt, because flaccid paralysis is not very uncommon in meningitis, especially if it is of the tuberculous type.

A positive diagnosis can only be made in most instances by lumbar puncture. The characteristics of the cerebrospinal fluid during the preparalytic stage have already been mentioned. During the acute stage it is clear, not infrequently under somewhat increased pressure, often forms a fibrin clot on standing and always contains an excess of protein and of cells. The cells are chiefly of the mononuclear type, most of them being lymphocytes. These characteristics are, however, the same as those found in tuberculous meningitis, the type of meningitis with which infantile paralysis is most likely to be confused. A positive diagnosis between them can only be made, therefore, by the examination of the cerebrospinal fluid for tubercle bacilli. The characteristics of the cerebrospinal fluid

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in meningococcal, influenzal and pneumococcal meningitis are so different from those in infantile paralysis that no confusion with these conditions is possible.

The Polyneuritic Type.—Pain is a prominent symptom in many instances. It is sometimes located in the joints, but is more often along nerve-trunks or indefinite in its distribution. It is usually most marked in the paralyzed parts. There is occasionally tenderness over the nerve-trunks. The pain and tenderness are sometimes marked enough to cause the paralysis to be entirely overlooked and a diagnosis of rheumatism or scurvy to be made.

The extremities are often held rigidly and all motion as vigorously resisted as possible, because of the pain on motion. This combination of rigidity and resistance is possible, of course, only when the muscles are partially paralyzed or when some of them are intact. The failure to appreciate the significance of this combination of flaccidity and spasticity not infrequently leads to errors in diagnosis during the acute stage.

PREVENTION

The question of the prevention of the disease is of great importance, and you are probably asked as frequently as we are in Massachusetts: "What shall I do to protect my children from the disease?" — a question which we must answer fairly with due regard to the interests of the patient, remembering, however, that advice too radical will increase the panic which is sure to appear in a locality where the disease is prevalent. It seems proper to advise people with children that, other things being equal, it is wise not to go for the summer to a locality where the disease is known to be prevalent. If, however, people find themselves in such a locality, the children and members of the family should keep away from other children who are in the acute stage of paralysis, and from children who are sick with other indefinite ailments.

In such localities it would seem wise to keep children away from children's entertainments, circuses and electric cars, and to prohibit playing with sick or paralyzed domestic animals.

Length of Quarantine.—We have at present no data as to how long the infectious period of the acute stage lasts, and any attempt to determine the length of quarantine must be mere guesswork.

With regard to the necessary length of quarantine, general consent has placed it at from two to four weeks. The recent experiments of

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Osgood and Lucas in this matter are, however, important inasmuch as they found an active virus in the nasopharyngeal mucous membrane of a paralyzed monkey, five and one-half months after the active stage of the disease had passed. The virus disappears from the nervous system in three or four weeks, or less, according to Flexner's experiments.

MORTALITY

The death-rate is about seven or eight per hundred, as the disease has existed in Massachusetts, but no foreign statistics show as low a figure as this, being from 10 to 22 per cent. In persons older than 10, the mortality-rate is higher than between 1 and 10, and under 1 year the disease seems more fatal.

The mortality table is interesting as showing the difference between the foreign and American death-rate.

TABLE 2.—COMPARISON OF FOREIGN AND AMERICAN DEATH-RATE

	Year	Cases	Death	Mortality Per cent.
Caverly, Vermont	1894	132	18	14.5
Wickman, Sweden	1905	868	145	16.7
Leegaard, Norway	1905	577	84	14.5
Zappert, Austria	1908	266	29	10.8
Lindner and Mally, Austria	1908	71	16	22.5
Furntrat, Steiermark	1908	433	57	13.1
Krause, Germany	1909	633	78	12.3
Muller, Germany	1909	100	16	16.0
Peiper, Germany	1909	51	6	11.7
Eichelberg, Germany	1909	34	7	20.6
Mass., U. S. A.	1907-1910	1,599	125	7.9

The mortality of cases in older children and in adults is higher than in other classes.

TABLE 3.—SHOWING HIGHER MORTALITY IN THE MORE ADVANCED AGES

	Age	Mortality, Per cent.
Wickman	12 to 32	27.6
Leegaard	15 to 30	25.8
Furntrat	Over 15	25.5
Lindner and Mally	Over 11	50.0
Mass., 1910	Over 10	20.0

PROGNOSIS

A few years ago it was supposed by most persons that practically all patients once paralyzed were to some extent paralyzed for life. But more

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careful study has shown us a better and more encouraging state of affairs. In the cases in Massachusetts, in 1909, a group of 150 occurring near Boston were studied in detail and it was reported that twenty-five, or 16.7 per cent., had wholly recovered inside of three months. This statement was not believed and the investigators were sent back to review their work. Each one of the children was stripped and the movements of ankle, knee, hip, shoulder, elbow, wrist and spine were examined. The statement can be definitely made, therefore, that, of this group, 16 per cent. had wholly recovered within three months.

The commonest type of final paralysis is an affection of one leg varying in degree from a slight limp in walking to a condition which requires the use of a brace; if both legs are involved, crutches are occasionally also required.

In short, a disability from infantile paralysis so great as to prevent an adult from earning his or her own living, occurs in a very small number of patients properly treated, but the ultimate condition of neglected cases is frequently distressing.

In connection with the question of treatment it is important to consider the reasons for this tendency to recovery, which are to be found in the pathology of the disease.

At the height of the disease there is an extensive perivascular infiltration of the blood-vessels of the cord, diminishing their lumen and inducing anemia and edema of the regions of the cord supplied by them. In addition to which there is also inflammatory edema of the nerve-tracts in the white matter and edema of the nerve-cells in addition to any destructive process which may be going on in neighboring cells. These changes, of course, cause loss of function in the parts affected, expressed as a temporary paralysis which passes away as the perivascular infiltration subsides. This apparently accounts for the improvement of paralysis in the early stage of the disease which exists in all non-fatal cases.

But the spontaneous improvement in the paralysis of the early history of the disease is no more important than improvement to be obtained by proper treatment in the chronic stage of the disease, after there is every reason to believe that the temporary perivascular infiltration has subsided, and the reason for this later improvement apparently rests on another pathologic basis. Assuming that the phenomena just described have ended, but that permanent destruction of certain motor centers in the

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spine has occurred, leaving a definite motor paralysis, the question arises, What reason is there to look for improvement in this? The answer apparently is as follows:

The spinal motor centers are grouped in cigar-shaped bundles whose length lies in the long axis of the spine. At each level of the spine are several of these bundles which overlap each other and communicate freely. Each bundle furnishes impulses to more than one muscle and each muscle receives impulses from more than one bundle. The blood-supply of the spine comes in horizontally and the lesions of this disease are most marked around the vessels. The distribution of the destructive process is therefore likely to be horizontal, while the distribution of the motor bundles is longitudinal. For this reason it is likely that if the destruction has not been too extensive, certain parts of the motor bundles will have escaped, leaving certain cells in some of them capable of function but which, in order to perform function, must acquire new connections and new groupings.

For this reason it will be seen, as will be maintained later, on the ground of practical experience, that muscle-training offers the best hope of the restoration of power to affected muscles in the chronic stage of the paralysis. It is an attempt to bring motor impulses from the brain down to the affected centers in the cord, there to establish new connections and new routes to the paralyzed muscles. The more impulses of this sort, however feeble they may be at first, to reach the affected muscles the better the chance of establishing finally routes which may serve for the transmission of impulses strong enough to secure efficient muscular contraction.

TREATMENT — ACUTE STAGE

In the matter of treatment much progress has been made in the last four years. In the acute stage meddlesome and useless treatment by stimulating drugs and counter-irritants has become largely discredited. Rest is very important, although cathartics and hexamethylenamin are of probable value, the latter drug being used with the object of setting free formaldehyd in the cerebrospinal fluid. Indeed, it has been shown experimentally that if hexamethylenamin is administered by mouth to monkeys, and if such animals are subsequently inoculated either subdurally or by the intracerebral route, paralysis is, in a certain proportion of cases, delayed or prevented. The value of the drug, therefore, in the prevention of infection would seem to be indicated, but there is as yet

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no experimental evidence to show that the drug is of much use after the disease has once begun.

In view of the pathologic condition, it is simply common sense to keep the patient recumbent and quiet and to avoid irritation of the muscles supplied by the affected nerve-centers. Massage and electricity should not, therefore, be used until there is reason to believe that subsidence of the inflammation has taken place. We may probably interpret the tenderness of the limbs as showing, while it lasts, the persistence of some degree of spinal or neural inflammation and until that has wholly disappeared we are not justified in the use of passive movements, massage or electricity.

Some men of large experience discourage all active treatment before the expiration of six weeks at least, but excellent results are frequently obtained by beginning treatment earlier on cases characterized by slight and transient tenderness. We should, however, discourage all active treatment until tenderness has wholly disappeared.

Another point of primary importance in early treatment is the prevention of contractures and deformities due to unopposed muscular action. Except in paralysis of the back and shoulder these contractures and deformities are always preventable, the contracture of the tendo Achillis, e. g., which is so frequent and so troublesome, being easily prevented by the use of splints supporting the foot at a right angle.

We read much concerning the relative merits of electricity and massage in the restoration of muscular power, but who shall pronounce as to the value of either in an affection in which paralysis varies so much in extent and severity and the recovery from which is so surprisingly different, irrespective of methods of treatment? Both methods have as an object the stimulation of muscular fibers and the maintenance of muscular nutrition, but both are inferior to muscular training in active treatment. This method of treatment is most useful in connection with massage or electricity and aims to cultivate the transmission of a voluntary motor impulse from the brain, down the cord to the paralyzed, weakened or stretched muscles. A flicker of motion, in an affected muscle, is often the forerunner of a return of some degree of power, and assistance by the hand in performing voluntary movements of the foot, for example, is often of great use.

Apparatus.—As to the use of apparatus, when a child in attempting to stand cannot, for example, hold the knee extended in weight-bearing,

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or when he stands in a position of deformity, for instance, with flat foot, apparatus should be applied to restore normal relations. Nothing but harm can result from sitting or going about with a curved spine, a hyperextended knee, or a foot rolled over on its inner or outer border. Stretching of muscles and ligaments and permanent bony deformities which are sure to result are worse than the use of braces, objectionable as the latter are.

Contractures which exist in neglected cases are not extremely resistant early in their history and must be removed before satisfactory mechanical treatment can be carried out. Most of them can be removed if stretched by means of bed traction or by the use of a series of correcting plaster bandages or by cutting. Operative treatment, except for the minor tenotomies, should not be undertaken for two years after the attack and preferably not then in children under 10.

Surgical Treatment.—Operation.—Tendon transplantation is a most useful measure and now that it has passed through its period of extravagant claims and unjustified condemnation, it may be said, in properly selected cases and with proper technic, to produce generally satisfactory and often brilliant results.

It consists in transplanting the distal end of a healthy muscle to the periosteal insertion of a paralyzed one, the foot or knee or arm being thus rebalanced. Since we have learned to prolong tendons by silk, the range and application of the operation has increased. We plan our operation, dissect and cut off our good muscle and if it is too short lengthen it at its distal end by heavy silk, we then insert it where we like, a new and sound tendon forming around the silk. In this way peroneals may replace tibials at the ankle or *vice versa,* hamstrings may be carried forward and inserted into the tibia to serve as knee extensors when the quadriceps is paralyzed and the trapezius may be substituted at the shoulder for a paralyzed deltoid.

Arthrodesis.—As a means of stiffening and making stable useless joints, arthrodesis has been found a useful measure. This operation consists in opening such joints and removing enough cartilage to secure ankylosis, and is applicable to the hip, ankle, elbow, and shoulder in children over 12, and to the knee in adults but not to the knee in growing children.

Silk Ligaments.—For the correction of foot drop in paralyzed legs we have made use more and more of anterior silk ligaments, the idea being

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to prevent plantar flexion of the foot but still to allow dorsal flexion. Heavy strands of silk are sewed into the anterior surface of the tibia, carried down under the annular ligament of the ankle and sewed into the dorsal surface of one of the tarsal bones. This operation is distinctly preferable to arthrodesis.

Nerve transplantation has not yet emerged from its experimental stage.

Finally, one of the most important things that orthopedic surgeons have learned of late years is that the hope for improvement in these cases through efficient and suitable treatment need not be given up in six months or one year or ten years, but that the possibility of improvement seems to persist almost indefinitely.

III. INFANTILE PARALYSIS AS OBSERVED IN MASSACHUSETTS, 1907-1910

The investigation of infantile paralysis in the state of Massachusetts by the State Board of Health for the four years, 1907 to 1910, inclusive, shows that during that period 2,138 cases of the disease were reported to the office of the State Board of Health. These cases were distributed by years as follows:

1907	234
1908	136
1909	923
1910	845

It is apparent from these figures that the disease in Massachusetts has, in a general way, during the period mentioned, increased in frequency, and it has been shown that a larger number of towns were infected each year.

Two-Year Periodicity.—There is, moreover, in these figures a suggestion of what may be an important phenomenon, and that is the appearance of a two-year periodicity for this disease. This phenomenon is noticeable in the distribution in general. In 1905 occurred the great Scandinavian epidemic. In 1906 there were no important epidemics. In 1907 occurred the New York epidemic. In 1908 there were only a few small epidemics and none of importance reported in Europe. In 1909 occurred the Massachusetts, Minnesota and Nebraska epidemics, two large and one small German epidemics, a large outbreak in Austria and the epidemic in Cuba. In 1910 from Europe no large epidemics

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were reported so far as we have been able to learn. The occurrence of the disease in this country in 1910 has already been dealt with.

This two-year periodicity has been noted a number of times in our investigation; for instance, in 1907 the western part of the state was quite sharply affected with infantile paralysis, whereas in 1908 the disease was almost non-existent in that same region. Again in 1909 the disease cropped up, but in 1910 hardly any evidence of infantile paralysis was noted in that neighborhood. In 1908, furthermore, in the Turners Falls district, etc., a very marked epidemic occurred, involving sixty-nine cases in a quite sharply circumscribed locality. In 1909 but one case occurred in this whole region. So that one can prophesy, to a certain extent, at least, that a limited locality, severely affected during one year, will probably enjoy partial immunity in the succeeding year. On the other hand, many localities having the disease to a moderate extent seem to go on from year to year at about the same rate.

Relative Prevalence in Smaller Cities and Towns.—Infantile paralysis is a disease affecting by preference the smaller cities and towns. This statement rests on the observation of 2,138 cases in Massachusetts, which have been analyzed in this regard.

The incidence per 1,000 of the population was recorded for every affected city and town in the state for four years. For each year the cities and towns were then arranged in the order of greatest incidence and then the first twenty-five on the list were contrasted with the last twenty-five for each year.

The average population for the first twenty-five was then reckoned for the four years and proved to be 5,205. The average population of the last twenty-five on the list, those where the incidence was least, was 52,674. A further study of the table shows that some such proportion as this existed in every one of the four years. That is to say, cities and towns where the disease was relatively least frequent were ten times as large on the average as those where it was most frequent. Furthermore, as a control, we tabulated the cases of scarlet fever reported in the state for the year 1910 in the same way. In the twenty-five cities and towns in which scarlet fever was most prevalent the average population was 6,446, and in the twenty-five where it was least prevalent per 1,000 of the population the average population was 7,633, practically the same, showing that scarlet fever showed no such predilection for the smaller cities and towns (Table 4).

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TABLE 4.—COMPARATIVE PREVALENCE OF INFANTILE PARALYSIS AND SCARLET FEVER WITH REFERENCE TO POPULATION OF CITIES

Disease	Twenty-five Cities Where Disease was *Most* Prevalent	Twenty-five Cities Where Disease was *Least* Prevalent
Acute epidemic poliomyelitis	5,205	52,674
Scarlet fever	6,446	7,633

In seeking to analyze some of the conditions which were constant in the smaller cities and towns which did not prevail in the larger, the number of domestic animals in each city and town was looked into and the analysis contributes the information that the number of such animals is relatively very much greater in the smaller cities and towns than in the larger. Such a fact may have no significance whatever, or it may in the future prove of importance (Table 5).

TABLE 5.—PREVALENCE OF INFANTILE PARALYSIS WITH REFERENCE TO THE NUMBER OF ANIMALS IN CITIES AND TOWNS

Total Number of Animals Assessed in Twenty-five Cities and Towns where the Disease was *Least* Prevalent, i. e., in the larger Cities and Towns.		Total Number of Animals Assessed in the Twenty-five Cities and Towns where the Disease was *Most* Prevalent, i. e. in the smaller Cities and Towns.	
Total population	943,614	Total population	142,639
Total number of cows	11,160	Total number of cows	12,801
1 cow to 84.5 inhabitants.		1 cow to 11 inhabitants.	
Total number of horses	29,519	Total number of horses	10,352
1 horse to 32 inhabitants.		1 horse to 14 inhabitants.	
Total number of swine	3,288	Total number of swine	2,374
1 swine to 287 inhabitants.		1 swine to 60.5 inhabitants.	
Total number of fowls	41,887	Total number of fowls	79,797
1 fowl to 22.5 inhabitants.		1 fowl to 1.75 inhabitants.	
Total number of dogs	23,854	Total number of dogs	5,865
1 dog to 39.5 inhabitants.		1 dog to 24.3 inhabitants.	

Of course, this is only one of many conditions which are different in the two classes but the persistence year after year of this greater prevalence of the disease in the smaller cities and towns must mean something with regard to the etiology of the disease if we were only able to interpret it.

Seasonal Occurrence.—The occurrence of the disease by months shows that the maximum incidence of infantile paralysis occurs between June

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and November. In 1907 the largest number of cases occurred in September. In 1908 July and August had equal numbers; 1909 had the largest number in August, and 1910 in July. There seems thus to be in Massachusetts a distinct tendency of the maximum incidence to recede in point of time, a phenomenon which should it occur in 1911, might be worthy of serious investigation (see Charts 5, 6, 7, 8 and 9).

Age.—The combined experience of three years, 1908 to 1910, shows that 6.13 per cent. of the cases occurred before the end of the first year; 66.94 per cent. before the end of the fifth year, and 83.1 per cent. before the end of the tenth year. Moreover, the Massachusetts figures give the impression that the disease is more frequent in adults than has been commonly supposed (Table 6).

TABLE 6.—OCCURRENCE BY AGE PERIODS (1908-1910)

Age, Years	Cases	Per cent.
Birth to 12 mos., inc.	55	6.13
1	119	13.26
2	155	17.35
3	125	13.93
4	92	10.25
5	54	6.02
	600	65.94
6 to 10, inc.	145	16.16
	745	83.10
11 to 20, inc.	83	9.25
	828	92.3
21 to 30, inc.	39	4.34
31 to 72, inc.	17	1.89
Not stated	13	.....
	897	.....

Sex.—As far as sex is concerned, the relative incidence is expressed by the figures of 56 per cent. for males and 44 per cent. for females, as reported for the four years, 1907 to 1910 (Table 7).

TABLE 7.—SEX (1907-1910)

		Per cent.
Males	906	56.66
Females	689	43.08
Not stated	4	0.25
Total	1,599	.....

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Mortality.—The mortality under 1 year is high, being for 1909-10 12.19 per cent. Between 1 and 10 years the fatality seems to drop, being 6.24 per cent., but after ten years the mortality again rises and reaches 14.81 per cent. The average mortality is 7.9 per cent. (Table 8).

TABLE 8.—MORTALITY BY AGE (1909-1910)

Age, Years	Cases	Deaths	Mortality, Per cent.
Under 1	82	10	12.19
1 to 10	945	59	6.24
Over 10	189	28	14.81
Totals	1,216	97	.....
Average mortality			7.9

Environment of Patients.—As regards the character of the house, practically an equal number of cases occurred in detached houses, in tenements or in apartments. Furthermore, there seemed to be no marked predilection of the disease for any special floor of the house. The house was said to be dry in 72 per cent., damp in 27 per cent., and medium in 1 per cent. No apparent relation to moisture can, therefore, be made out. The sanitary conditions were either excellent, good or fair in 85.6 per cent. cases, and bad in only 11.5 per cent. The age of the house was not remarkable, 41.14 per cent. of patients living in new houses; that is to say, within the first and tenth year of its existence; whereas 58.85 per cent. lived in houses more than ten years old.

It has seemed at times as if the disease preferred river beds, but this factor in the occurrence of the disease must rest on other basis than mere propinquity, because, although thirty-three patients are said to have lived near a river or near water, 317 lived more than one-eighth of a mile from any stream, pond, or beach, so that it seems probable that any marked occurrence of the disease along the water courses is due to other factors than a neighborhood of water, the determining influence being in all probability increased personal contact due to the fact that all highroads, electric railwavs and railroads are very apt to follow river valleys, and cities and towns are, therefore, more likely also to be situated along such water courses.

In the same way, no especial relation could be found between the occurrence of infantile paralysis and nearness to railroads. The experience of Wickman, as shown by his maps, certainly seemed to show a

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distinct preference of the disease for localities near railroads, but in Massachusetts we have not been able to observe any etiologic connection between the two. In 1909-10, 30.56 per cent. of the cases occurred within one-eighth of a mile of the tracks, while 69.42 per cent. were more than one-fourth of a mile away.

A study of sewage condition shows that the disease occurs under all conditions of sewage disposal and as regards water supplies from metropolitan and city systems, as well as from wells and springs (Tables 9, 10, 11, 12, 13, 14, 15, 16 and 17).

TABLE 9.—CHARACTER OF HOUSE (1907-1909)

		Per cent.
Detached	220	49.54
Tenements	224	50.45
Total	444	.....

TABLE 10.—FLOOR OF HOUSE INHABITED BY FAMILY (1907 AND 1909)

		Per cent.
The whole house	75	21.06
The first floor	139	39.04
First floor and basement	3	.84
Second floor	101	28.37
Third floor	32	8.98
Upper stories	6	1.68
Total	356	.....

TABLE 11.—LOCATION OF HOUSE (1907-1910)

		Per cent.
Dry	459	71.38
Damp	177	27.52
Medium	7	1.08
Total	643	.....

TABLE 12.—SANITARY CONDITION (1907-1910)

		Per cent.
Excellent	123	18.83
Good	271	41.50
Fair	165	25.25
Bad	74	11.33
Not stated	20	3.06
Total	653	.....

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TABLE 13.—AGE OF HOUSE (1909-1910)

		Per cent.
New House (1-10 yrs.)	144	41.14
Old House (over 10 yrs.)	206	58.85
Total	350	.....

TABLE 14.—NEARNESS OF HOUSE TO WATER (STREAM, POND OR BEACH) (1909-1910)

		Per cent.
On river	33	9.42
Within ⅛ mile	66	18.85
Within ¼ mile	89	25.42
Within ½ mile	78	22.28
Within 1 mile	84	24.00
Total	350	.....

TABLE 15.—NEARNESS OF HOUSE TO RAILROAD (1909-1910)

		Per cent.
On railroad	57	16.28
Within ⅛ mile	50	14.28
Within ¼ mile	44	12.57
Within ½ mile	114	32.57
Within 1 mile	85	24.28
Total	350	.....

TABLE 16.—CHARACTER OF SEWAGE DISPOSAL (1908-1910)

		Per cent.
Sewage (Met.)	110	26.25
Sewage (City, etc.)	211	50.35
Cesspool	23	5.48
Vault	60	14.31
Privy	15	3.57
Total	419	.....

TABLE 17.—CHARACTER OF WATER-SUPPLY (1908-1910)

		Per cent.
Metropolitan	120	27.98
Town	62	14.45
City	172	40.09
Well	17	3.96
Spring	58	13.51
Total	429	.....

Rainfall.—The deficiency in rainfall during the last eight years has been, of course, very marked, and this factor may be of importance in the spread of the disease. It certainly has to be taken very seriously into consideration if we accept the suggestion of Hill of Minnesota that dust,

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especially dust infected with horse manure, may be responsible for the transference of this disease. The deficiency of rainfall in Massachusetts has varied since 1904, when it was 1.35 inches, to 9.42 inches in 1910. With the deficiency of rainfall during each year of this period has occurred a sort of cumulative effect, which makes this deficiency relatively more important each year. A study of the deficiency of rainfall shows that the largest number of cases have not occurred in the driest years in Massachusetts (Table 18 and Charts 10, 11, 12, 13).

TABLE 18.—DEFICIENCY RAINFALL (1904-1910)

Year	Cases in State	Actual	Normal	Deficiency
1904	...	43.81	45.16	— 1.35
1905	...	37.60	.....	— 7.56
1906	...	43.21	.....	— 1.95
1907	234	44.49	.....	— 0.67
1908	136	37.61	.....	— 7.55
1909	923	42.10	.....	— 3.06
1910	845	35.50	.....	— 9.42
				—31.56

Dust.—A study of the amount of dust in affected localities shows a report of a moderate amount in more than half of the cases while in 36 per cent. of the total number much dust or an excessive amount was reported. The matter is not, however, one capable of measurement or exact determination and the table cannot be regarded as important (Table 19).

TABLE 19.—RELATION TO DUST (1909-1910)

		Per cent.
No dust	0	.....
Very little dust	29	8.28
Moderate amount of dust	195	55.71
Much dust	118	33.71
Excessive amount of dust	8	2.28
Total	350	.....

Insects, Etc.—The possible transfer of the disease by vermin, insects, rodents, etc., was investigated, but the figures are not at all conclusive, and very little knowledge of value is likely to come in this department of the work, until individual infected localities are investigated by persons

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expert in the study of entomology, a line of investigation of the greatest importance (Table 20).

TABLE 20.—PRESENCE OF VERMIN, INSECTS, RODENTS, ETC. (1909-1910)*

		Per cent.
Flies	298	90.85
Mosquitoes	142	43.29
Mice (house)	123	37.5
Ants	97	29.57
Rats	77	23.47
Bedbugs	70	21.34
Roaches	64	19.51
Spiders	54	16.46
Mice (field)	24	7.31
Biting flies	10	3.04
Fleas	4	1.21
Grubs and Caterpillar	3	.91
Bees	2	.609
Sand fleas	1	.304
Snakes	1	.304
Crickets	1	.304
Squirrel	1	.304
Leeches	1	.304
Brown tail moths	1	.304
Moles	1	.304
Total	328 families	

*Report 1907 and 1908 only for flies and mosquitoes.

Domestic Animals.—The rôle played by domestic animals in the transfer of the disease is one of great possible interest, but as yet the interrelation of the paralyses of man with those of animals has not been sufficiently marked to justify any definite conclusion. Many cases of paralysis in animals have been reported, but the instances in which paralysis in human beings has been coincident with such animal paralysis have been rare (Table 21).

TABLE 21.—DATA AS TO DOMESTIC ANIMALS (1909-1910)

		Per cent.
No animals of any kind in	131	39.93
Animals in	197	60.06
125 animals had sickness, paralysis or death in	70	21.34
Total	328 families	

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Transmissibility.—The degree of contagiousness of infantile paralysis is a question of great theoretic and practical importance. It is one which at the present time it is practically impossible to analyze exactly because, associated with the paralytic cases of this disease, occur an unknown number of so-called abortive cases which may be of the greatest importance in transferring to others paralytic types of the affection. Furthermore, these unknown abortive cases become immune to subsequent infection, and if included in any study of comparative contagiousness, will go far, undoubtedly, to vitiate any conclusions drawn. Our figures show that in 350 cases carefully studied, there was direct contact with an acute case in 13 per cent.; direct contact with possible abortive cases in 1.42 per cent.; with chronic cases in 4.28 per cent.; and that there was, undoubtedly, indirect contact with an acute case through a third person in 4 per cent. of the cases (Tables 22 and 23. See Chart 14 for an endemic focus in a city of 119,000 inhabitants).

TABLE 22.—INSTANCES OF CONTAGIOUSNESS (1909-1910)

Certain direct contact:		Per cent.
With acute case	46	13.14
With possible abortive case	5	1.42
With chronic case	15	4.28
With both acute and chronic cases	2	.57
Certain indirect contact with acute case by third person	14	4.00
Number of cases investigated	350	.....

TABLE 23.—FAMILIES WITH MORE THAN ONE CASE (REASON FOR SUSPECTING CONTAGION) 1908-1910

		Per cent.
One case	357	92.24
Two cases	27	6.97
Three cases	3	.77
Total	387	.....

Institution Cases.—Forty-four institutions for children in Massachusetts were written to in 1910, in order to ascertain whether or not any cases of acute epidemic poliomyelitis had originated within those institutions during the year. One institution, containing 360 children, in Fall River, reported a single case, a boy, 4 years old. No secondary cases occurred in this institution (Table 24).

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TABLE 24.—INSTITUTION CASES	Cases	Incidence per 1,000
44 institutions with 3,600 children under 15 years of age	1	.277
Mass., with a population of 851,092 children under 15 years (Census of 1905) showed in 1910 cases of acute epidemic poliomyelitis under 15 years	532	.625

Children in institutions therefore would seem to be, in some measure and in some way as yet unexplained, protected from this disease. Their opportunities for contact with the outside world are obviously much curtailed in comparison with those of the ordinary child, and therein lies very probably the reason for their immunity.

Swimming, Etc.—Just prior to the disease, there was a history of swimming, wading, or paddling, in water more or less contaminated, in 25 per cent. of the cases just before attack (Table 25).

TABLE 25.—SWIMMING, WADING OR PADDLING (1908-1910)*

		Per cent.
Swimming, wading or paddling	105	25.05
No such history	314	74.94
Total	419	.....

*Water contaminated more or less in every instance.

Accidents.—In the same way, 34 per cent. of 457 cases gave a history of accidents, over-exertion or falls. With regard to the swimming, etc., the percentage would seem no higher than one would expect in any group of children at this season, living near the water. But the percentage of thirty-four accidents, falls and over-exertion just before the attack would seem perhaps unduly high (Table 26).

TABLE 26.—ACCIDENT, OVER-EXERTION OR FALL PRECEDING ATTACK (1907-1910)

		Per cent.
Accident	41	8.97
Over-exertion	47	10.28
Fall	67	14.66
No such history	302	66.08
Total	457	.....

Other Diseases.—A study of the disease prevalent in affected towns coincident with infantile paralysis showed nothing of importance. A further study of recent antecedent illnesses in the patient showed merely a number of minor ailments of no especial significance (Table 27).

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TABLE 27.—DISEASES PREVALENT IN TOWN AT TIME OF OCCURRENCE OF INFANTILE PARALYSIS (1909-1910)

	Cases	Per cent.
Not known	159	46.49
Infantile paralysis	120	35.08
Whooping cough	12	3.508
Measles	11	3.21
Gastro-enteritis	8	2.33
Diarrhea	6	1.75
Influenza	6	1.75
Tonsillitis	4	1.16
"Rheumatic fever"	4	1.16
Scarlet fever	3	.87
Mumps	2	.58
Malaria	2	.58
Pneumonia	1	.29
Coryza	1	.29
Chicken pox	1	.29
Typhoid	1	.29
Diphtheria	1	.29
Total	342	.....

Diet. — The relation of diet to infantile paralysis is possibly of importance, but as yet no definite relation between the disease and certain kinds of food has ever been made out. Although evidently unusual, the disease does occur in breast-fed babies, four cases of the kind having been recorded in our returns of the last two years (Table 28).

TABLE 28.—DIET (1909-1910)

		Per cent.
General	283	80.85
(a) Raw cow's milk	308	88.00
(b) Condensed milk	15	4.28
(c) Breast milk and other food	19	5.42
(d) Breast milk alone	4	1.14
Fish	265	75.71
Fruit	288	82.28
Berries	197	56.28
Meat	275	78.57
Canned goods; (a) fruits; (b) vegetables; (c) fish	202	57.71
Cereals	140	40.00
Bread and butter	23	6.57
Vegetables	275	78.57
Stews and soups	11	3.14
Eggs	186	53.14
Tea, coffee, cocoa	6	1.71
Malted milk	2	.57
Ice cream	83	23.71
Predigested and beef juices	4	1.14
Candy	27	7.71
Bananas	6	1.71
For cases numbering	350	.....

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Symptoms.—Digestive disturbances are common before, during and after the attack. Diarrhea is less frequent, comparatively, than constipation (Table 29).

TABLE 29.—DETAILS OF DIGESTIVE DISTURBANCE (1909-1910)

		Per cent.
PRECEDING ATTACK		
Nausea and vomiting	120	34.28
Constipation	109	31.14
Colic	5	1.42
Diarrhea	39	11.14
Indigestion or stomach upset	4	1.14
Mucus in stools	2	.57
ACCOMPANYING ATTACK		
Nausea and vomiting	140	40.00
Constipation	149	42.57
Colic	15	4.28
Diarrhea	56	16.00
Indigestion (indefinite)	3	.85
Mucus in stools	1	.28
FOLLOWING ATTACK		
Nausea and vomiting	5	1.42
Constipation	122	34.85
Colic	2	.57
Diarrhea	18	5.14
Mucus in stools	2	.57
Data for cases numbering	350	.....

The analysis of the general symptoms of the acute attack in 720 cases show fever present in 88 per cent., brain symptoms in 37 per cent., and retraction of the head in 34 per cent. Sore throat was only recorded as present in 9 per cent. (Table 30).

TABLE 30.—GENERAL FEATURES OF ACUTE ATTACK (1907-1910)

		Per cent.
Fever	636	88.33
Pain and tenderness	528	73.33
Brain symptoms	266	36.94
Retraction	248	34.44
Sore throat	65	9.02
For cases numbering	720	.....

Appearance of Paralysis.—The appearance of paralysis in relation to the attack was studied in 589 cases. In 16 per cent. paralysis occurred on the day of the attack; in 16 per cent., one day after the attack; in

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17 per cent., two days after the attack and in 16 per cent., three days after the attack. That is to say, in 65 per cent., within three days after the attack. In two cases it was recorded as coming before the attack and in twenty-three cases (4 per cent.) between one and two weeks after the onset (Table 31).

Pain and Tenderness.—The great importance of pain and tenderness as a symptom is not sufficiently recognized; 1,198 cases were analyzed in this regard and in 70.5 per cent. these symptoms were present. The disappearance of pain and tenderness in an analysis of 604 cases, in which the time was recorded, occurred in 30 per cent. within one week, in 15 per cent. in from one to two weeks, and in 13 per cent. in from two to four weeks. In five cases it was recorded as lasting two to three months and in three cases as lasting several months (Tables 32 and 33).

TABLE 31.—APPEARANCE OF PARALYSIS IN DAYS AND WEEKS AFTER ONSET OF FEVER (1907-1910)

		Per cent.
Preceding attack	2	.33
Same day	95	16.12
One day	93	15.78
Two days	103	17.49
Three days	98	16.63
Four days	58	9.84
Five days	22	3.73
Six days	51	8.65
Seven days	18	3.05
Eight days	6	1.01
Nine days	2	.33
Ten days	4	.67
Eleven days	3	.509
Twelve days	5	.84
Thirteen days	1	.169
Fourteen days	4	.67
Not known (fatal)	1	.169
2 to 3 weeks	6	1.01
3 to 4 weeks	1	.169
4 to 5 weeks	1	.169
8 weeks	1	.169
Two days previous	1	.169
Not stated	13	2.207
Total	589	.....

TABLE 32.—PAIN AND TENDERNESS (1907-1910)

		Per cent.
No pain	178	14.85
Pain	845	70.53
Not stated	175	14.6
Total	1,198	.....

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TABLE 33.—DISAPPEARANCE OF PAIN AND TENDERNESS (1909-1910)

		Per cent.
1 day or less	11	1.82
2 days	22	3.64
3 days	29	4.801
4 days	15	2.48
5 days	14	2.31
6 days	3	.496
A few days	28	4.63
1 week	59	9.76
1-2 weeks	91	15.06
2-3 weeks	46	7.61
3-4 weeks	33	5.46
4-5 weeks	1	.16
6-7 weeks	1	.16
1-2 months	28	4.63
2-3 months	5	.82
Several months	3	.496
Until death	39	6.45
Present when report was made	175	29.13
Total	604	.....

Distribution of Paralysis.—The distribution of paralysis was studied in 1,158 cases. The most common location was in one leg, occurring in 28 per cent.; both legs came next in frequency with 24 per cent.; one or both arms in 9 per cent.; and where one arm and leg were affected, a hemiplegic distribution was more common than a crossed paralysis, the relative percentages being 10 per cent. and 3 per cent. Special attention should be called to the frequent occurrence of paralysis of the back, abdomen, neck and face, which are much more frequent than is supposed and too often overlooked (Table 34).

TABLE 34.—DISTRIBUTION OF PARALYSIS (1907-1910)

		Per cent.
One leg only	324	27.97
Both legs only	272	23.48
Back	154	13.29
Both arms and both legs	129	11.13
One arm and leg, same side	110	9.49
One arm only	84	7.25
Both legs and one arm	75	6.47
Face	74	6.38
Abdomen	67	5.78
One arm and leg, opposite sides	33	2.84
Respiration	31	2.67
Both arms only	23	1.98
Neck	11	.94
Both arms and one leg	10	.86
Deglutition	7	.604
Neck and back	6	.51
Ataxia (transitory)	5	.43
General	3	.25
Intercostal	1	.086
Both arms, back, chest and throat	1	.086
For cases numbering	1,158	.....

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Prognosis.—In order to throw additional light on the question of prognosis, Dr. B. E. Wood of Boston was requested by the Board to investigate the present condition of all patients reported to the Board as paralyzed in the year 1907. This inquiry was made with especial reference to the number of recoveries occurring in the four years which had elapsed. Letters were sent to all physicians who had reported cases in 1907, asking whether or not such patients had recovered, and when recovery was reported, such cases were seen unless they had disappeared or moved, and if such was the case all available information was obtained about the patients.

Of 234 cases, twenty-two could not be investigated, eleven had died in the acute stage of the disease; eight others had died subsequently of other diseases. Although two of them are said to have recovered from the paralysis, no trace could be found of three cases. In 212 cases, therefore, the termination is known, and fifty-ceven of these (27 per cent.) are reported as having recovered.

On investigation it was found that there were three classes of recovery: (1) Complete recovery without atrophy, (2) recovery with complete function but with some atrophy, (3) recovery with some hypertrophy of the affected limb. The table shows the proportion of each of these.

TABLE 35.—CONDITION OF PATIENTS AFTER RECOVERY

		Per cent.
Complete recovery without atrophy	16	28.1
Functional recovery with atrophy	21	36.8
Recovery with some hypertrophy	3	5.3
Recovery, presence or absence of atrophy unknown	17	29.8

When atrophy was present, the maximum amount noted was as follows:

TABLE 36.—REMAINING ATROPHY AFTER RECOVERY

Calf	1¾ inches
Thigh	1¼ inches
Arm	½ inch
Forearm	¾ inch

In most cases the atrophy was much less than this, a difference of one-eighth inch being counted as atrophy, but in all such cases function of every muscle was perfect. The amounts of hypertrophy recorded were in two instances one-fourth inch and in one, three-eighths inch, all of the calf.

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In these cases the severity of the attack was classed as:

TABLE 37.—SEVERITY OF THE ATTACK

		Per cent.
Severe	14	24.5
Moderate	9	15.9
Mild	28	49.1
Not noted	6	....
	57	....

The distribution of the paralysis in the fifty-seven recovered cases was as follows:

TABLE 38.—DISTRIBUTION OF THE PARALYSIS

One lower extremity	12
Both lower extremities	9
One upper extremity	6
Both upper extremities	2
One lower and one upper extremity	10
Three extremities	7
Four extremities	5
Face alone	1
General	1
Not given	4

In addition to this distribution, paralysis of the face also existed in four, of the back in five, of the face and back in one, and of the abdominal muscles in one.

The time of recovery in the fifty-seven cases was as follows:

TABLE 39.—TIME OF RECOVERY

1 week or less	2
1 week to 1 month	8
1-2 months	8
2-3 months	5
3-6 months	10
6-12 months	9
1-2 years	5
2-3 years	5
No data	5

CONCLUSIONS AS TO PROGNOSIS

The following conclusions seem justified as regards prognosis:

1. Complete recovery or functional recovery occurs in something over 25 per cent. of patients examined at the end of four years.

2. Atrophy may exist without impairment of function.

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3. In about half of the recovered patients the onset was mild.

4. The distribution of the paralysis in such cases was not essentially different from that of cases which do not recover.

5. The period of recovery in many instances occupied months, and in several cases from one to two years.

234 Marlboro Street—145 State House.

ABSTRACT OF DISCUSSION

ON PAPERS OF DIXON AND KARSNER AND LOVETT AND RICHARDSON

DR. PHILIP KING BROWN, San Francisco: I shall take up the subject of poliomyelitis from a different point of view, one that concerns us more particularly in the West. I have been associated with three epidemics of this disease, two of which are not on record. Besides these there have been two others in this state. In 1898, during the month of June, Dr. Newmark and I studied an epidemic of four cases that occurred in the San Joaquin Valley in the town of La Grand, with a population of but forty-nine people. One of these patients died. In 1903 Dr. Alice Wood reported twenty-five cases in and about San Francisco. In 1906 I collected eight cases in an epidemic at Watsonville, Cal., most of them seen by Dr. Saxton Pope. None of the patients died. Last year I collected twenty-five cases within a radius of a few miles at the upper end of the Sacramento Valley. Drs. Lennon, Fleischner and others collected in the neighborhood of seventy-five cases in San Francisco and vicinity last winter, and a report of this epidemic will be made next month by Dr. Fleischner.

The importance of this historically is the thing I want to mention. We have seen in California epidemics of poliomyelitis in towns quite apart, one of them far away from railway communications, two in a great center and one in a thickly-settled agricultural district. Some of these epidemics occurred in the fall and winter during the rainy season and some in the summer months when no rain falls in California. Often there is cesspool drainage in country districts and pollution of the wells occurs. We must all appreciate that this disease occurs in epidemics and that it bears a definite relation in some way to bacterial growth.

The facts that we must reconcile to-day are those presented by the readers of the first paper and those in the investigations reported from Massachusetts. The latter, together with what Dr. Simon Flexner has reported thus far, have enabled us to secure definite information regarding the type of the disease met with and certain facts regarding the organism responsible for the disease. It is quite possible that the organism referred to by the reader of the first paper may be shown to be responsible for the disease, but the failure of a large number of investigators to obtain corroborating proof must make us suspend judgment, and indicates plainly how difficult the task is of proving the connection of an organism with this disease. Flexner and Lewis, Gay and Lucas, and many others in this country and abroad, have given us valuable data about the characters of the infecting organism but no conclusive facts as to the organism itself.

The fact that we are dealing with disease at times acutely epidemic should be clear when we read the report by Müller of an undoubted epidemic that occurred in Nauru, an island in Oceania, where there appeared 700 cases among 25,000 inhabitants. He states that in three months 650 showed no paralysis whatever. That fact indicates the varying intensity of the disease as it occurs in different

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epidemics, for our experience in this country indicates a resulting paralysis as high as 90 per cent. in some epidemics. Doubtless Müller was able to include the abortive cases in his group, whereas they must frequently escape notice in epidemics in cities or less detached localities.

What all of us are interested in is this: It is a type of disease that is not recognized as a rule when it occurs sporadically, until the paralysis occurs. This is told us by men with an abundant experience but it should not deter us from being on the lookout for the disease at all times all over the country. The chief value of this symposium seems to me to be the knowledge of the multiform character of the disease and the help in recognizing it and dealing with it, which becomes now our common property through the efforts of these investigators.

In several cases that appeared during an epidemic in Northern California the febrile conditions lasted for ten days. I saw several patients who were brought to San Francisco still in the febrile stage. The reason for keeping the children quiet is because the destructive trouble is chiefly in the cervical and lumbar regions. The process, of course, is a diffuse myelitis and the thickest parts of the cord suffer most, probably from mechanical conditions. Activity could only increase the evil effects of these conditions.

At the last meeting of the Association of American Physicians at Atlantic City, the report of Dr. Simon Flexner of the Rockefeller Institute for Medical Research was of great interest, particularly as to what he stated with regard to the use of hexamethylenamin (urotropin) and certain allied products. He also calls attention to the great desirability of keeping the nasal mucous membrane cleansed with some mild antiseptic solution. The result is very efficacious in preventing the microorganisms from penetrating the mucous membrane or from being transmitted to others through breathing, sneezing, or coughing.

The question of diagnosis is so complicated that I do not feel like considering it; but I will say this: one must look out for evidences of the disease and one must be constantly prepared to meet it. The occurrence of coryza, acute bronchitis with fever, pain in the neck, and other early symptoms of this disease should make us careful. When such an instance is met with, we should insist on keeping the child in bed and under careful observation.

Caverly, in an epidemic that occurred in Vermont, referred to the appearance of the disease in animals. A spinal cord of a chicken was sent to Dr. Charles Dana, who satisfied himself that there was the same form of degeneration in it that occurred in the human being.

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